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# H.R. 1961—VIETNAM VETERANS AGENT ORANGE RELIEF ACT

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HEARINGS
BEFORE THE
SUBCOMMITTEE ON
COMPENSATION, PENSION, AND INSURANCE
OF THE
COMMITTEE ON VETERANS' AFFAIRS
HOUSE OF REPRESENTATIVES
NINETY-EIGHTH CONGRESS
FIRST SESSION

APRIL 26, AND APRIL 27, 1983

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# H.R. 1961, VIETNAM VETERANS AGENT ORANGE RELIEF ACT

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## TUESDAY, APRIL 26, 1983

House of Representatives,
Committee on Veterans' Affairs,
Subcommittee on Compensation, Pension and Insurance,
*Washington, D.C.*

The subcommittee met, pursuant to notice, at 9 a.m., in room 334, Cannon House Office Building, Hon. Douglas Applegate (chairman of the subcommittee) presiding.

Present: Representatives Applegate, Hall, Daschle, Montgomery, Mica, and McEwen.

Also present: Representative Harrison.

### OPENING STATEMENT OF CHAIRMAN APPLEGATE

Mr. **Applegate**. Good morning. The subcommittee will come to order.

This morning we start a series of hearings on H.R. 1961,[1] a bill introduced by our colleague, Mr. Daschle of South Dakota. The bill would provide service-connected compensation and other ancillary benefits to veterans who served in South Vietnam on the basis of their possible exposure to the herbicide agent orange. The bill is narrow in scope in that it addresses three specific disabilities, soft tissue sarcoma, porphyria cutanea tarda, which is a liver condition, and for the skin condition of chloracne.

The bill contains no time limit within which these disabilities must be first shown to exist, but it does contain a sunset clause that would terminate benefits so granted a year after the study mandated by Public Law 96-151 is submitted to the Congress.

We will first hear from Mr. Daschle, the sponsor of the bill. Following Mr. Daschle's testimony, we will hear from our colleague from Michigan, the Honorable Dave Bonior, to be followed by the Administrator of Veterans' Affairs, the Honorable Harry Walters. We will conclude today's meeting by hearing from the commander in chief of the Veterans of Foreign Wars and representatives from a number of other veterans' organizations.

Tomorrow we will hear from Ms. Barbara Boxer, Congresswoman from California; Dr. Donald Custis, Chief Medical Director of the Veterans' Administration; and Miss Dorothy Starbuck, Chief Benefits Director of the agency. In addition, we will hear from a number of professional and technical witnesses.

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[1] See p. 161.

In our deliberations on this bill, the committee will face, for the first time, the issue of granting benefits simply on the possibility that there may be a basis for authorizing compensation. I think it is important for us to keep an open mind as we sit and listen to the various witnesses today and tomorrow and other testimony during these long deliberations. I think it is going to be very important that we all keep our minds open and study this testimony.

There are a number of studies currently underway in addition to the Centers for Disease Control study mandated by Public Law 96-151. I ask unanimous consent that a listing of these studies be included in the appendix of this hearing record.

[The information appears on p. 173.]

Mr. APPLEGATE. The results from some of these studies are expected to be available later this year.

Some of the literature on earlier studies on the possible harmful effect of herbicides seem to suggest that persons who are significantly exposed to herbicides suffer a higher incidence of soft tissue sarcoma than would be expected among the general population. There also seems to be a consensus of opinion, although not unanimous, that this is not true. In an effort to get at the truth, or as near to that goal as possible, the Congress passed Public Law 96-151 mandating one of the most massive epidemiological studies ever undertaken. The Centers for Disease Control in Atlanta has started this project which is estimated to cost the Veterans' Administration somewhere between $40 million and $100 million.

Because the findings from the CDC study are not expected to be available until 1987, the sponsors of H.R. 1961 are proposing that compensation be paid and the full range of service-connected benefits be made available for certain specific disabilities while the study is ongoing.

At this time, I will recognize our very distinguished chairman of the full committee, Mr. Montgomery, for any remarks that he may want to make.

## STATEMENT OF HON. G. V. "SONNY" MONTGOMERY, A REPRESENTATIVE IN CONGRESS FROM THE STATE OF MISSISSIPPI

Mr. MONTGOMERY. Thank you, Mr. Chairman. I want to commend you and the other members of the subcommittee for having these hearings. This probably is one of the most important pieces of legislation that will come before the House Veterans' Affairs Committee this year.

I certainly agree with your statement that we should keep an open mind until we have heard all of the witnesses and the testimony that will take place this week, and then exercise our best judgment. It is precedent-setting, in effect, pertaining to agent orange and presumptive findings, but this is a burning issue with the Vietnam era veteran, and I understand it. Therefore, we certainly have to be fair and provide a full hearing, which you are about to do.

Thank you very much, Mr. Chairman.

Mr. APPLEGATE. Thank you, Mr. Chairman.

Today we will be operating under the 10-minute rule for witnesses, and the 5-minute rule for questions. I would ask that all witnesses cooperate to the maximum degree possible. Full statements of all witnesses will be included in the hearing record. It is going to be somewhat of a long day, and it could drag on if we allow this to get too far out of hand. So we would, again, ask for your cooperation.

At this time, I recognize our colleague on the committee and the chief sponsor of H.R. 1961, Mr. Tom Daschle of South Dakota. Tom, you may speak from the table, if you prefer. We are looking forward to your testimony.

## STATEMENT OF HON. THOMAS A. DASCHLE, A REPRESENTATIVE IN CONGRESS FROM THE STATE OF SOUTH DAKOTA

Mr. DASCHLE. Thank you, Mr. Chairman.

I use this forum because I have a very special interest in this particular issue, and I want to address the committee first as a witness.

I believe, really, that this committee and the Congress and the American people, at long last, are making progress on the agent orange issue. I commend this committee and our chairman for taking an active involvement in finding out the ramifications that we talk about today.

For the first time, we are addressing legislation which would compensate certain Vietnam veterans suffering ill health as a result of their exposure to toxic herbicides, including agent orange, which contain dioxin. You are to be commended for allowing this first step to be taken.

It is my firm belief, however, that we wouldn't even be here today, that we wouldn't have heard and continue to hear from thousands of sick and dying Vietnam veterans, if the Dow Chemical Co. had made a good-faith effort to tell this Government, principally the USDA and the Department of Defense, of their knowledge and concern about dioxin's toxicity in 1965 when they decided to convene chemical industry representatives for a meeting on the subject. As we have recently learned, Dow executives ignored the warning from their own researchers. Now, for more than 15 years, veterans and others exposed to dioxin have been suffering.

It is clear that Dow has waged a concerted effort to withhold documents pertaining to dioxin's effects, and it is equally clear that this information would be invaluable to this subcommittee in pursuing a legislative remedy for thousands of Vietnam veterans who are presently suffering. I, therefore, propose that this subcommittee subpoena from Dow all information pertaining to the 1965 meeting of chemical industry representatives, as well as any and all research Dow has now conducted on dioxin.

But regardless, Mr. Chairman, of culpability, we have an obligation to provide for those who serve this Nation in wartime and who are now sick and dying.

This committee has shown compassion and deep understanding to those veterans who have lost their limbs, their eyes, or the use of their bodies.

This committee has shown determination in fighting for VA facilities to care for these brave people. We must always do so.

But times are changing. New wars have brought new wounds. Modern conflict has brought about change in the way we fight. And now it must also bring about change in the way we treat our veterans.

H.R. 1961 is a conservative beginning in our acknowledgement of that fact. It will compensate veterans suffering from soft tissue cancer or a liver disease known as porphyria cutanea tarda, or chloracne.

We can be quite detached from all of this unless we take the time to learn of these diseases and what they do.

Many are dreadful—devastating. They destroy the body and, in doing so, sometimes the mind. They have to represent the closest thing to hell that we can find here on Earth. You only have to look at a victim's agonizing eyes or listen to his story—and then later on hear of his death.

When that happens, you change. You lose that detachment and you develop a deep commitment to find out how you can help these people. You want to cry out, "Why? Why does it have to happen at all? Why haven't we helped these people, especially as U.S. veterans? Why?"

Once we were told it was cost. That is the reason. Yet, this bill will cost the U.S. Treasury $4 million annually. The 5-year cost of H.R. 1961 will be less than what is spent each day—each day—on food stamps and less than what is spent every 3 months on military bands.

Culpability, that is another reason. The Government does not want to show culpability, we are told. Yet, this Government paid $33 million to buy out Times Beach, Mo., several months ago, even though it was a private individual who poisoned the community in the first place. An Illinois jury was convinced enough of dioxin's harm to award 47 railroad workers $58 million.

The third reason, the one most often given, is the lack of complete certainty that exposure to dioxin is the cause of these diseases. This is the flimsiest reason of all. We all know that exposure causes these diseases in animals when tested, but fortunately we don't test humans. We all know that there are many studies which do show the same effect on humans.

We all know that there are veterans who served in Vietnam who were exposed to dioxin and who now have these diseases. Yet, this Government still refuses to pay compensation or to offset their complete loss of income and perhaps their death.

Sure we must continue to study the scientific questions here. Yes, we have to be skeptical of drawing premature conclusions.

But look upon H.R. 1961 then as an interim measure. Support my proposal to sunset these benefits if the CDC studies show that there is no link between dioxin and these medical illnesses.

Consider what happens if we fail to act. Look one of these cancer victims in the eye and tell him that he will have to wait. Or imagine your feeling upon learning of conclusive evidence that dioxin exposure does cause cancer if for 15 to 20 long years we have said "no" to our veterans who lie dying while imploring our help.

There is no good reason not to act. We owe these veterans the same presumption we have already given victims of at least 40 other illnesses written into law.

So let us act now out of ample precedent, out of ample evidence, and out of our continuing determination to send a clear message to our past and future veterans that this country will care for its wounded regardless of war and what new warfare may bring.

At long last, let us tell those veterans, those veterans who are patiently waiting, that meaningful help is on the way.

Thank you, Mr. Chairman.

Mr. APPLEGATE. Thank you, Mr. Daschle. Thank you for a very sensitive yet tough statement.

Mr. MONTGOMERY. Mr. Chairman, I just want to commend the gentleman. We have been working for years together on different areas of concern to the Vietnam veteran. He and Mr. Bonior have headed up the caucus in the Congress working for the Vietnam era veteran, and Mr. Daschle has always cooperated with the chairman.

We will certainly take a good, sincere, hard look at this bill. We will give it our full intent to see where the problems are and what can be worked out.

Mr. DASCHLE. Thank you, Mr. Chairman.

Mr. APPLEGATE. The gentleman from Texas, Mr. Hall.

Mr. HALL. I have no questions, Mr. Chairman.

Mr. APPLEGATE. Again, thank you very much, Mr. Daschle.

Our next witness will be the Congressman from Michigan, Mr. Dave Bonior. Dave, your complete statement will be entered in the record, and you may proceed as you wish.

## STATEMENT OF HON. DAVID E. BONIOR, A REPRESENTATIVE IN CONGRESS FROM THE STATE OF MICHIGAN

Mr. BONIOR. Thank you, Mr. Chairman.

Mr. Chairman, I am honored to have the opportunity to appear again before this committee and address the agent orange issue. This committee has played a vital role in agent orange policy throughout these many years.

Its leadership has helped force an aggressive research agenda on an all too often hesitant Veterans' Administration. Two years ago, as you know, in a landmark effort, this committee provided interim eligibility for health care for Vietnam veterans with agent orange related problems.

Enactment of H.R. 1961 is, I believe, consistent with this tradition of leadership. I want to commend the present chairman of the Vietnam Veterans in Congress, Mr. Daschle, the chairman of this subcommittee, Mr. Applegate, and the chairman of the Subcommittee on Hospitals and Health Care, Mr. Edgar, for their role in the development of this legislation, which is now cosponsored by more than 160 Members of the House of Representatives.

Mr. Chairman, over 3 years ago, two independent Swedish epidemiological studies related exposure of 2,4,5-T, a main ingredient in agent orange, to soft tissue sarcoma. Despite the fact that the studies were in the public domain, indeed, were part of the Environmental Protection Agency's case against 2,4,5-T, the Veterans' Administration did not even know of their existence.

Yet, the VA was not moved by the evidence nor embarrassed by their ignorance. In an almost apparent rush to dismiss the facts, a scant 12 days after receiving the studies, the VA Administrator at that time concluded:

> * * * following preliminary analysis, my scientific advisers have informed me that they do not think the papers make a major contribution to answering the problem that is of concern to us. I have been advised that these studies appear to be based on a random distribution.

Not everyone agreed with the VA assessment. Some 3 months later, the distinguished Office of Technology Assessment and the Interagency Work Group on Phenoxy Herbicides completed their own review of the two studies. In the words of one OTA examiner:

> The studies are among the most carefully conducted investigations of their type that I have ever seen. In toto, the Swedish work is credible, if not fully conclusive.

The evidence has continued to mount. Over 1 year ago, an independent review of four additional groups of exposed American workers found the same correlation. Nearly 1 year ago, in the distinguished New England Journal of Medicine, two doctors from Emory University reported on three Vietnam veterans with soft tissue sarcomas.

Yet, even today, the VA has not yet been moved to provide compensation. Vietnam veterans have waited long enough for the unmovable VA to be touched by shame, if not compassion. H.R. 1961 is a responsible attempt to give Vietnam veterans a clear sign that Congress, at least, is prepared to act.

H.R. 1961 is not the hurried answer to a new problem. It does not open the floodgate to future claims, nor does it reject science in the name of compassion. Rather, H.R. 1961 is the considered product of long years of frustration.

At a yearly cost of just $4 million, its presumptions are carefully focused—very carefully focused, as the chairman of this subcommittee mentioned—on three disabilities—soft tissue sarcomas, chloracne and porphyria. In each case, substantial evidence exists relating the disabilities to exposure to 2,4,5-T in humans.

In considering H.R. 1961 now, this committee would not be prejudging the outcome of the epidemiological studies it has worked so hard to establish. H.R. 1961 creates only an interim eligibility that expires following the first major report on the epidemiological study mandated by Public Law 96-151. The committee's hands are free to adjust presumptions to the new evidence when it arrives.

Nor should the committee feel hesitant about acting in advance of special studies. Our knowlege of 2,4,5-T and dioxin did not begin with the Vietnam war and is not bound by the limits of studies focused specifically on Vietnam veterans. There is an ever growing body of independent animal and human evidence. Agent orange policy can and must respond to this material, as well as to the special studies.

H.R. 1961 will provide compensation to some 3,000 Vietnam veterans. That is an important step, but the bill's significance goes beyond the aid it offers to individuals. By demonstrating the Congress willingness to act now, H.R. 1961 will continue the effort begun by this committee to restore credibility to agent orange policy and its scientific agenda.

The credibility of the Government's research agenda does not rest exclusively on the quality of its science. It also rests on the integrity of the policy it serves. For Vietnam veterans to trust the call for more research, they must believe that new findings will produce new policy and will not disappear behind an ever escalating burden of proof and the call for yet more research. They must believe that, at some point enough will be enough, at some point a line will be drawn.

Reasonable people can differ over where the line should be drawn, but they cannot differ over the fact that each day, as claim after claim is denied, a line is being drawn in the lives of our Nation's Vietnam veterans. It is possible to argue that the evidence supporting the disabilities included in H.R. 1961 is not adequate, but it is not possible to avoid the question that must follow upon that argument. If this evidence is not adequate, then how tall a mountain of material will finally be required?

Vietnam veterans cannot be asked to believe in science for science's sake. They must be asked to believe in science in service to a responsible but compassionate policy. H.R. 1961 provides that policy. It demonstrates that the Congress is prepared to draw a line and act.

Mr. Chairman, with your permission, I would like to insert in the record a copy of the two Swedish studies, the OTA and Work Group reviews of those studies, and the subsequent observations noting a relationship between exposure to 2,4,5-T and soft tissue sarcomas.[1]

I thank you, and I would be happy to answer any questions that you or your colleagues may have.

[The statement of Mr. Bonior appears on p. 185.]

Mr. APPLEGATE. Without objection, that will be included in the record, Mr. Bonior.

David, thank you very much for your appearance here today. We appreciate it very much. We are aware of the lead which you and Tom Daschle have taken in bringing this legislation before the committee. You are to be commended for your efforts.

The gentleman from Ohio, Mr. McEwen.

## STATEMENT OF HON. BOB McEWEN, A REPRESENTATIVE IN CONGRESS FROM THE STATE OF OHIO

Mr. McEWEN. Thank you, Mr. Chairman.

We meet today to consider legislation concerning perhaps the most perplexing issue to confront our subcommittee, our full committee, the House of Representatives, and, indeed, all veterans during this session of the U.S. Congress, the 98th session.

Mr. Chairman, the issue of granting service-connected compensation on a presumptive basis to Vietnam veterans who were exposed to agent orange with the three disabilities mentioned in H.R. 1961, a bill introduced by our colleague, Mr. Daschle, contains just about every element of possible consideration associated with the passage of legislation. These elements are medical in nature, they are scientific in nature, they are statistical in nature, and presumptive in nature. Moreover, they involve resolving reasonable doubt in favor of veterans.

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[1] See pp. 196, 203.

Mr. Chairman, it is fair to say that they are also very emotional. It is also fair to say that they are argumentive. All of good will and good intentions will also differ on the conclusions reached here.

So, Mr. Chairman, today we will hear our colleagues, Mr. Daschle and Mr. Bonior, differ with the Administrator of Veterans' Affairs. We will hear the national commander of the Veterans of Foreign Wars testify that he supports presumptions called for in H.R. 1961. His statement also eloquently argues for liberalization of the criteria for granting service connection to the veterans involved. But in a spirit of complete fairness, he also states:

> I am not here to argue the scientific merits or demerits of the case for compensating Vietnam veterans due to herbicide exposure-related health problems. There are more than enough scientists available for such an exercise.

The American Legion will also testify in favor of the bill before us. Its representative will state that dioxin exposure may result in the three disabilities in question, and they will share some scientific information with us that tends, in their view, to support their position. They conclude by stating that they appreciate that the intent of this legislation must undergo the prudent deliberation of this subcommittee. The legion then states that, "We have no doubt that H.R. 1961 will be closely, but objectively, scrutinized."

The Disabled American Veterans and the Paralyzed Veterans of America both come before the committee often as very effective spokesmen for disabled veterans. They will oppose the bill. The DAV will say that "our track record will speak for itself" and, Mr. Chairman, I believe that we can all agree with that assertion. DAV's representative recognizes H.R. 1961 as a sincere effort to make up for lost time on this issue, but one that brings into serious question the possible impact it may have upon the integrity of the VA's disability compensation program which it states has served the Nation well for more than 60 years. In DAV's view, H.R. 1961 deviates substantially from traditional methods of establishing a factual basis upon which to pay compensation for disability.

The Paralyzed Veterans of America, Mr. Chairman, also gives reasons for its position on H.R. 1961. Its position is that, while it applauds the basic efforts of those of us who are sponsors, it does not, in their opinion, meet the test of presumption which it says must be related to sound, reasonable, medical evidence.

Mr. Chairman, we will also hear testimony from the Veterans of the Vietnam War. I am sure that it will be very moving. The National Veterans Law Center will present a statement that is in the nature of a legal brief and an argument. It contains a number of important statements and conclusions.

Finally, Mr. Chairman, on tomorrow, we will receive expert testimony from the scientific community and the Veterans' Administration.

Mr. Chairman, many of us in the Congress have an open and very inquisitive mind on this issue. We want to do what is right. We want to hear all of the witnesses, and we want to fully digest their testimony. When it is over, Mr. Chairman, each of us on this subcommittee will make the best informed judgment that we can. If the evidence is there, I will be for H.R. 1961 and, hence, my cosponsorship. I will resolve any questions of a legal nature or in a moral nature in favor of the Vietnam veterans exposed to agent orange. I will do what is expected as a member of this subcommittee. Less would be unconscionable; more than this, I do not believe is expected.

Thank you very much, Mr. Chairman.

Mr. APPLEGATE. Thank you, Mr. McEwen.

The gentleman from Texas, Mr. Hall.

Mr. HALL. I have no questions, Mr. Chairman.

Mr. APPLEGATE. Mr. Daschle.

Mr. DASCHLE. Mr. Chairman, I just want to applaud Mr. Bonior's statement and also Mr. McEwen's. Mr. Bonior, in particular, has been a leader in this effort, as you said when you introduced him. He has been one of those in the Vietnam Veterans Caucus that has always been able to rally support. As a member of the Rules Committee, in particular, I have a special interest in your role in Congress this year. So I want to assure you that we are going to do everything we can to work with the committee and to insure that, at some point this year, we can take this legislation to the Members of the House of Representatives.

So I thank you again, Dave, for an excellent statement.

Mr. BONIOR. Thank you for your kind remarks.

Mr. APPLEGATE. The gentleman from Mississippi.

Mr. MONTGOMERY. Thank you, Mr. Chairman.

I join my colleagues in commending Mr. Bonior for his very strong statement. He has been very active in the Vietnam movement, along with Mr. Daschle and others.

I only have one question, Dave, and maybe Mr. Daschle can comment on it also. As I understand the bill that you have cosponsored, Mr. Daschle's bill, H.R. 1961, there is a sunset provision or a closeout after 5 years if this legislation is enacted until the study is completed; is that correct?

Mr. BONIOR. There is a sunset provision; that is correct, Mr. Chairman.

We recognize that there are two ongoing specific studies that have been mandated, the ranch hand study and the study that you had a hand in in moving to Atlanta—and I appreciate that—which was mandated by Public Law 96-151.

The problem, of course, with those two studies is that, in terms of the ranch hand study, we are not going to have enough statistical evidence to determine the mortality rates for probably at least a half a decade and probably more in terms of a decade. With respect to the other issue, the epidemiological study mandated by Public Law 96-151 that is ongoing in Atlanta, Ga., at the Centers for Disease Control, that is a much longer study that will take some time to determine. The question then becomes, What do you do in the meantime with respect to these people who have these serious problems?

So we do have, as you have indicated, a sunset provision in the law. But we believe that the evidence is conclusive right now with respect to the soft tissue sarcoma, the chloracne, and the porphyria.

I have put forward for the record a number of studies done by, I might add, very eminent people, Dr. Axelson and Dr. Hardell, two of the people who did these Swedish studies, are considered tops in their field in the world in this type of work. We have also included some other reports in the medical journals, two specific medical journals.

But to answer your question, yes; we recognize that there are ongoing studies that Congress has mandated and, indeed, if it should so happen, and we all hope it does, that these studies indicate that there are not serious health problems that warrant these compensations, then corrections can be made.

Mr. **Montgomery**. Thank you.

Mr. **Applegate**. Thank you, Mr. Chairman.

Mr. **Daschle**. Mr. Chairman, could I followup with one additional question?

Mr. **Applegate**. Certainly, Mr. Daschle.

Mr. **Daschle**. In my statement, Dave, I made a suggestion or a proposal to the subcommittee. When the chairman addressed the question of the need for additional information, it brought it to mind.

I made the proposal that we subpena information from Dow Chemical because, obviously, they are the ones who have done a tremendous amount of research, none of which has been made available to the committees of the Congress. What is your feeling about that information from the research you have done? Is that perhaps an important element that we are missing at this point?

Mr. **Bonior**. I think it is extremely important. I am very pleased that you have suggested that be done.

As you know, Dow has worked on this chemical and its cousins for a very, very long time, much earlier than, of course, Vietnam. The stuff was available commercially, I believe, in the 1960's if not sooner. I am not sure if it goes beyond the 1960's. It would be of extreme help to this committee in understanding this issue if we could have that evidence. I would hope that the committee would act positively on that.

Another reason for that, I might add just parenthetically, is that there has been some suggestion that there has been a serious problem about the Midland plants in Midland, Mich., especially for women who are pregnant, because of dioxin exposure. It would be interesting for this committee to take a look at that particular evidence and studies that are ongoing in that area.

Mr. **Applegate**. Thank you very much, Mr. Bonior.

Mr. **Bonior**. Thank you.

Mr. **Applegate**. We have with us this morning the Honorable Harry Walters, the Administrator of the Veterans' Administration, who has asked for some time to make some comments on behalf of the agency.

Tomorrow, we will hear from Veterans' Administration witnesses, including professional and program directors, who will comment at length on the bill.

Mr. Walters.

## STATEMENT OF HARRY WALTERS, ADMINISTRATOR, VETERANS' ADMINISTRATION

Mr. **Walters**. Thank you, Mr. Chairman. It is a pleasure to be there today to address the subcommittee.

I am here today to state the Veterans' Administration's policy concerning the complex, far-reaching issues raised by the use of agent orange in Vietnam. As you know, the Chief Benefits Director, the Chief Medical Director, and other major representatives of the Veterans' Administration will, pursuant to the committee's request, present testimony as to the specific issues tomorrow.

I would also like to express my appreciation to Mr. Bob Currieo, the commander in chief of the Veterans of Foreign Wars, for extending to me the courtesy of yielding a few minutes of his time so that I may make the following statement. As per our agreement, Mr. Chairman, to enable Mr. Currieo to depart here on schedule, I will be pleased to respond to any questions. submitted to me for the record rather than respond at this time.

The use of agent orange in Vietnam has caused much concern and given rise to controversy. Although we are here today to discuss a proposal of presumptive law for purposes of compensation, the issues of herbicide exposure and its possible effects are in fact of much broader and worldwide concern. The overriding issue is whether herbicide exposure has long-term effects on the health of, not only Vietnam veterans, but of all humans.

At my confirmation hearing, I pledged that I would be the advocate for America's veterans. I promised to administer the laws enacted on their behalf fairly, effectively, and compassionately. I have and will continue to do all that I can to insure that Vietnam veterans who may have been exposed to phenoxy herbicides receive health care for disabilities that could possibly be associated with such exposure in accordance with the provisions of Public Law 97-72 and other laws. I view my responsibilities and obligations as a sacred trust.

I fully appreciate the time it is taking to perform the medical research and obtain the medical evidence essential to a resolution of these critical issues. I can assure you that I am committed to actively, fully, and enthusiastically supporting the numerous medical studies now underway or planned which will determine whether exposure has long-term health consequences.

Since January 1 of this year, the Veterans' Administration has, among other things, undertaken to improve its agent orange registry, we have expanded its efforts to provide information concerning agent orange to veterans and other concerned parties. We have transferred by interagency agreement the performance of the epidemiological study and the supporting resources to the Centers for Disease Control. We have reorganized and revitalized the chloracne task force. We have improved the communications and coordination between key officials in the VA, most notably our agent orange projects office, and our field personnel.

In addition, the following studies are underway: One, a Vietnam veterans identical twin study; two, Vietnam veterans mortality study; three, with the Environmental Protection Agency, a retrospective dioxim/furan adipose tissue study; four, update of the analysis of the worldwide literature on herbicides; five, sponsoring the authorship of a monograph series by internationally recognized experts; and six, continuing to solicitations of additional specific research projects from VA scientists.

The Air Force is completing the ranch hand study, the results of which should be available in June of this year. Further medical research and other studies regarding the possible health effects of herbicide exposure have been and are being conducted, both within the United States and around the world. The VA has responded to the controversies raised by the Swedish papers suggesting dioxin as a possible causative factor of soft tissue sarcoma.

The Veterans' Administration with the Armed Forces Institute of Pathology has begun an epidemiological study of soft tissue sarcoma. The Centers for Disease Control has announced its intention to conduct a similar epidemiological study. Public health authorities in the States of New York, Washington, and Kansas have likewise announced studies of soft tissue sarcoma.

The endeavors of the Veterans' Administration and others to obtain the medical evidence necessary to resolve these complex issues is now broad based and is ongoing. It is my hope that the discussions which follow today and in the months ahead focus on the issues. The VA is now attempting to search for the final medical conclusions to the complex agent orange exposure issue. It is my desire that this agency stand as a beacon of hope in its search for the final and conclusive answers and not, please, not as a whipping post for the debate.

The medical community has reached a consensus that a causal relationship exists between exposure to dioxins and chloracne, a skin disorder that appears within a relatively short period after exposure. The medical community has also reached a consensus that, in some individuals, porphyria cutanea tarda, a liver disorder, can be triggered by dioxin exposure. However, no medical studies have been concluded which establish a causal connection between exposure and the initial occurrence of these disabilities long after exposure. Accordingly, the Veterans' Administration has recognized both chloracne and PCT as compensable disabilities attributable to service connection.

Presently there is not a consensus within the medical community that a causal connection between phenoxy herbicide exposure and long-term health consequences in humans exists. There are several studies which, on a correlative basis, suggest that exposure to dioxin and/or phenoxy herbicides may be related to soft tissue sarcomas. There are many other studies that do not substantiate the suggested relationship. To date, there are only theories and hypotheses.

H.R. 1961 is based on these theories and hypotheses. It endeavors to presume that which is not as yet medically accepted or proven. H.R. 1961 is not based on evidence accepted by the medical community as reliable; it is not based on fact. Given the far-reaching consequences of the public policy addressed in part in H.R. 1961, I do not believe that Congress should base its decisions on hypotheses.

I, as Administrator of Veterans' Affairs, have an obligation to safeguard this Nation's compensation program for service-disabled veterans and the survivors of those who gave their lives for their country. The purpose of this program is to compensate individuals for injuries or diseases contracted in or aggravated by military service. The existence of a causal connection between disabilities and military service to the Nation is the fundamental precept of the compensation system. It is the basis of its integrity and its acceptability. Should H.R. 1961 become law, the basic premise of the program would be fundamentally changed and its continued viability jeopardized.

I must also express my concern with H.R. 1961's potential for alarming millions of veterans and the population as a whole. Most people will not understand the concept of presumption as expressed in H.R. 1961, that is, not based on acceptance by the medical community of a connection between exposure to herbicides and the occurrence of serious illnesses long after exposure. Rather, there is a serious risk that the message conveyed will be that herbicide exposure has in fact been medically accepted as causing, among other things, cancer. We must remember that herbicide exposure is of a worldwide concern.

As Administrator of Veterans' Affairs, I am committed to the rapid resolution of the agent orange issue. If a consensus of the medical community finds with reasonable medical certainty that exposure causes disabilities, I will insure that our veterans are fairly compensated for these disabilities. The Veterans' Administration will, of course, continue to provide health care to those veterans who may have been exposed to phenoxy herbicides for disabilities that could possibly be associated with such exposure.

Thank you, Mr. Chairman and members of the subcommittee, for your continued support of the Veterans' Administration in its efforts within present law to serve our Nation's veterans. Thank you.

[The statement of Administrator Walters appears on p. 230.]

Mr. **APPLEGATE**. Thank you, Mr. Walters.

By agreement, then, because of time, we will be submitting questions to you for your response.[1]

Mr. **MONTGOMERY**. Mr. Chairman, could we take about 5 minutes? I think it is very important—this is the head of the Veterans' Administration—maybe to put something on record. It is your subcommittee, but I am a little concerned. I am not sure whether he is for or against the bill. I would like to have that on the record. It is your subcommittee, but I am a little concerned, unless you could get him back.

Mr. **APPLEGATE**. I appreciate that very much, Mr. Chairman.

By agreement—and I feel I do have a commitment to stick to that agreement—we will be submitting questions for the Administrator to answer. I do anticipate that I will be asking Mr. Walters to return before the committee in the near future.

Mr. **DASCHLE**. Mr. Chairman?

Mr. **APPLEGATE**. Mr. Daschle.

Mr. **DASCHLE**. I will respect your decision, certainly, but I, like the chairman, would certainly like the chance to press Mr. Walters for a number of questions. Hopefully, we can bring him back.

I think that, with a statement like this, I would want to leave quickly, too. But I think that there are some things that we have to address.

There is one in particular that, as he is leaving, perhaps we can bring up. The Congress mandated that the Veterans' Administration Administrator conduct a scientific study to determine if there is a relationship between the amputation of an extremity and the subsequent development of cardiovascular disorder.

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[1] See pp. 236, 250.

There was one study which was made, and that study was made by the medical followup agency of the National Academy of Sciences. That study concluded that veterans who suffered an amputation of both legs or one leg above the knee would have a significantly higher degree of risk from dying from a disease of cardiovascular problems. On the basis of that one study, this administration provided disability compensation.

Now we are told that it is going to take 6, 7, perhaps 10 studies to provide the same kind of compensation opportunities for those afflicted with agent orange. I think that is the kind of questioning we would like to bring before the VA at some point in the very near future.

Mr. APPLEGATE. With your agreement, Mr. Daschle, and with that of Mr. Walters—which he has already agreed to do—we will have him come back and sit before the committee again.

Mr. MICA. Mr. Chairman?

Mr. APPLEGATE. Mr. Mica from Florida.

Mr. MICA. I also would like to ask some questions, but, in deference to your agreement, I have 10 questions that I would like to submit for the record and have the responses as quickly as possible,[1] and associate myself with the comments made by Mr. Daschle.

Thank you.

Mr. APPLEGATE. Thank you, Mr. Mica.

Thank you, Mr. Walters.

Mr. APPLEGATE. Our next witness will be Mr. James Currieo, commander in chief of the Veterans of Foreign Wars of the United States.

Mr. Commander, we would appreciate it if you would please introduce those are accompanying you here today.

## STATEMENT OF JAMES R. CURRIEO, COMMANDER IN CHIEF, VETERANS OF FOREIGN WARS OF THE UNITED STATES, ACCOMPANIED BY COOPER T. HOLT, EXECUTIVE DIRECTOR, WASHINGTON OFFICE, VETERANS OF FOREIGN WARS, AND PHILIP MAYO, ASSISTANT LEGISLATIVE DIRECTOR, VETERANS OF FOREIGN WARS

Mr. CURRIEO. Mr. Chairman, I would like to, first of all, introduce the two colleagues at the table with me. I have with me the executive director of the Washington office, Cooper T. Holt; and Mr. Phil Mayo, our assistant legislative director.

Also, if I may, before I begin my testimony, I had, on approval of your committee, to request that two guests be present with me here. As I travel throughout the country, I meet hundreds of veterans of the Vietnam war, and many are afflicted in one form or another. One of the questions that comes to me many times from those who have never met anyone afflicted by a possible condition related to dioxin poisoning—I felt it was necessary at least to have someone available to be here if any members of the committee wished to asked questions of them. I have two gentlemen with me this morning.

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[1] See p. 236.

I have Mr. Pete Murphy, a retired Air Force enlisted man who served in the military police, served with nuclear weapons and worked in Vietnam in Cam Ranh Bay, and also near Saigon in Nha Trang. He is suffering from a disability of the central nervous system. Last month, he had an agent orange examination by the Veterans' Administration and presently has a claim pending.

The other gentleman is Mr. Billy Housewright, who served in Vietnam in 1970, and was discharged in 1971. He is also suffering from a condition that could possibly be related to dioxin poison. He has a severe loss of hair and skin rash that can readily be seen.

These two gentlemen are in the second row if any members of the committee wish to ask questions of them.

Also I would like to introduce Mr. Jim Roxby, who will be part of the testimony on his own representing the Veterans of the Vietnam War. He is the local post commander of his unit in Pennsylvania. He is available and will be testifying a little later on this morning.

Mr. Chairman and members of the subcommittee, on behalf of the 1.96 million members of the Veterans of Foreign Wars—which I might add that over a half a million of our membership are veterans of the Vietnam war—I welcome the opportunity to express our views to this distinguished subcommittee regarding H.R. 1961, a bill introduced by Hon. Thomas A. Daschle for the purpose of providing a presumption of service connection for the occurrence of certain diseases related to exposure to herbicides or other environmental hazards or conditions in veterans who served in Southeast Asia during the Vietnam war. I am pleased to note that more than 160 Members of the House of Representatives have chosen to cosponsor this measure. This support is greatly appreciated by our organization.

Three resolutions dealing with the herbicide issue were adopted by the voting delegates to our most recent national convention in Los Angeles, Calif. Resolution 607, entitled "Herbicide Exposure," mandates that the Veterans of Foreign Wars seek a rapid and accurate scientific investigation of all aspects of this issue and that the criteria for disposition of claims be liberalized. The purpose of the resolution is to discover how all the problems related to herbicide exposure may be resolved medically and that claims for compensation for them be reasonably resolved.

Resolution 623, entitled "Herbicide Fee-Basis Counseling," calls for genetic and psychological counseling to be provided to veterans who served in Vietnam and their spouses.

Resolution 761, entitled "Agent Orange," resolves that legislation providing for compensation and health care be supported, and that outreach campaigns concerning these programs be funded and encouraged at all levels of government. A copy of each of these resolutions, Mr. Chairman, is appended to my statement for your review.

Primarily in the last 5 years, Mr. Chairman, considerable national attention has been focused on agent orange, a defoliant used extensively in the Vietnam conflict. Since 1978, the Veterans' Administration has received complaints from veterans concerning various forms of cancer, liver ailments, skin lesions, birth defects, neurological disorders, or other disorders that they attribute to exposure to agent orange. We, as well as others, have suggested that other causes of health problems in Vietnam also be studied, such as agent blue, arsenical; dapsone, an antimalarial drug; aflatoxin, a toxin occurring in Southeast Asia produced rice; and, of course, drug and alcohol abuse.

During this same timeframe, Mr. Chairman, the division in the scientific community over this issue has continued. The administrative arm of the Government had to be forced by law into undertaking scientific studies. The General Accounting Office found mismanagement in the effort to get studies on this issue underway. Legislation was required before an adequate level of health care was provided. Arsenic compounds have been examined and known to medicine for approximately a century and, yet, this information has not found its way into a review like that provided for dioxins. And, finally, the questions raised by the Government's offer to buy the community of Times Beach, Mo., are very real to veterans of the Vietnam war.

Mr. Chairman, I am not here to argue the scientific merits or demerits of the case for compensating veterans due to herbicide exposure-related health problems. There are more than enough scientists available for such an exercise.

While I do not presume to speak for them, I note that many veterans organizations, in some form, have supported the premise on enactment of this legislation. I view it as a compassionate and responsible response on the part of the Government to veterans of the Vietnam war.

Mr. Chairman, typical of the questions and concerns that come to our headquarters in great numbers and with which I am personally greeted in my extensive travels throughout our great country are those sentiments that came to us in a letter early in April of this year from a Jacksonville, Fla., veteran.

The letter follows:

### AGENT ORANGE

> That title has been used to sell all types of newspapers and magazines from the supermarket to scientific journals. By now, most of us know that it contained dioxin, deadly in Missouri, but absolutely no threat to Vietnam veterans. Most of us also know that flight records exist that show when and where herbicides were sprayed and/or dumped, and where they were stored, shipped and moved, et cetera. How can we find out where and when we might have been exposed, or put to rest our fears, knowing that most likely we were not exposed. Where is the published report or chart detailing herbicide location and military unit locations in the same time period? We know it was spread in the delta and on trails and on base camp perimeters. But when? Some one knows. How can we find out?

Mr. Chairman, I view this subcommittee as a jury. Our Government has asked its Vietnam veterans to continue to bear the legacies of their honorable service. Our Government has advised its Vietnam veterans that more evidence is needed to resolve doubt. How much more evidence is needed before some of this doubt may be resolved in favor of the veterans?

Our Government has asked its Vietnam veterans to wait while studies can be done. How much longer is it necessary to wait? It is easy for those who are not affected to say wait.

The legislation before the subcommittee should be enacted. It represents a beginning of another aspect of healing the wounds of the Vietnam war. How long will the jury be out?

Mr. Chairman, without objection, I have a number of materials with me today that I ask to be part of the record of this hearing along with my statement. Copies of these documents were supplied to your subcommittee earlier.

I would like to be available now to answer questions, or the guests who are with me.

Mr. **Applegate**. Thank you, Mr. Commander, that will be made a part of the record.

[The statement of Mr. Currieo with attachments appear on p. 269.]

Mr. **Applegate**. I have noticed in resolution 761 adopted by your convention that it starts with the premise that the U.S. military personnel were grossly contaminated and are suffering exposure. To help us in our final deliberations, can you share with us any evidence that you feel you might have that would show a specific and definite link between contamination and health consequences?

Mr. **Mayo**. The Department of Defense spray records.

Mr. **Holt**. Those records, Mr. Chairman, indicate every place that supposedly herbicides of any type were sprayed, the date, and how much was sprayed.

Mr. **Applegate**. I thank you very much. I understand that that information is available to us.

Thank you very much, Mr. Currieo, for presenting a very well thought out and well articulated statement here today.

The gentleman from Ohio, Mr. McEwen.

Mr. **McEwen**. Thank you, Mr. Chairman. I, too, join in expressing my appreciation for your testimony this morning.

Mr. Currieo, you are familiar with the Ranch Hand study.

Mr. **Currieo**. I am to a point, sir.

Mr. **McEwen**. Would you comment upon that for us?

Mr. **Currieo**. My familiarity involves the fact that the Ranch Hand Study was made—I think it was about 1,200 veterans who were tested or examined for possible contamination or for some effects because they were handling agent orange and spraying on board airplanes, and so forth.

I haven't seen any conclusive report as of yet as to what the findings of this study may produce.

Mr. **McEwen**. Do you have any concerns about the manner in which it is being conducted?

Mr. **Currieo**. I personally don't feel that that study alone should be conclusive evidence whether or not the dioxins in agent orange are causing these disabilities. I think it is a beginning. Of course, the CDC study—I visited the CDC a couple of months ago and talked to the officials there, and they are going to find out some answers, but it still going to take a while.

It appeared to me in our organization that we could go back instead of going forward and conduct some type of research, in addition to Ranch Hand, that possibly were affected and try to draw some correlation in that manner.

Mr. McEwen. Mr. Currieo, I am going to pose a question to many of the witnesses who will be here relating to the question that the Veterans' Administration has raised about the presumption issue, whether or not there should be scientific proof or whether or not the Congress should take the latitude for a presumption in favor of the veteran on a particular matter. Their contention will be that this forbodes serious ill for the compensation program in general in the future. I would like to give each one of you who support this measure an opportunity to respond to that.

Mr. Currieo. We don't feel it is going to jeopardize the integrity of the other compensation programs. This is a complex issue, there is no question about it. We are happy that hearings are now being held on compensation. We hope that, in addition to the research that is going on now, that probably more research will be conducted to go back and try to draw a correlation in this.

But we firmly believe there are those who are hurting, badly hurting, and if we wait 10 years down the line, it is going to have a disastrous effect, not only on compensation, but I believe on the Nation in general if we find out in fact that the veteran is suffering from what was related to agent orange or other herbicide used in Southeast Asia.

Mr. McEwen. You have no concerns that such requests will proliferate into other areas?

Mr. Currieo. No. The bill itself is very limited, and I think that this is a beginning.

Mr. McEwen. Thank you very much.

Mr. Mayo. Mr. Chairman, if I may, the rating schedule, as I understand it, evolved over a long period of time. It wasn't something we started out with as it is now, and we have had it in time immemorial. It was something that evolved. This may be another part of its evolution.

Mr. McEwen. Perhaps my question is not that clear. As I understand the Veterans' Administration's concern, it is that it will not only proliferate into other areas of agent orange injuries, but across the board for other presumptions of illness.

Mr. Currieo. You are saying other than agent orange?

Mr. McEwen. Yes.

Mr. Currieo. I believe that, if that happens, the Congress and the Veterans' Administration will handle that problem when they come to it.

Mr. McEwen. Very good. Thank you.

Mr. Applegate. Thank you, Mr. McEwen.

The gentleman from Texas, Mr. Hall.

Mr. Hall. Thank you, Mr. Chairman.

Mr. Commander, I have one or two questions that are raised in my mind by your testimony.

On page 1, you quote a resolution, No. 607, that was mandated when the VFW met in California a few weeks ago. You state the criteria for the disposition of claims should be liberalized.

On page 3, you say that the legislation before the subcommittee should be enacted because it represents the beginning of another aspect of the healing process for Vietnam veterans.

On page 2, you say that, during this timeframe, the division in the scientific community over this issue has continued. And then you further say at the bottom of page 2, "I am not here to argue the scientific merits or demerits of the case for compensating Vietnam veterans," and so forth.

The bill says on page 2, the one that you are asking us to pass—let me say at this time that there is no one on this panel who has any more compassion for Vietnam veterans than the gentleman from Texas, or veterans from any war for that matter.

On line 9 of this bill on page 2, you are asking that the Congress find "* * * defects occurring among the children of such persons, may be the result of the exposure of such persons during such service. * * *"

Over on page 3, line 13, subsection (2)(A), "* * * such disease shall be considered to have occurred in or aggravated by such service, notwithstanding that there is no record of evidence of such disease during the period of service."

Let's assume for a moment that this case of a veteran, whoever that veteran, may be, is submitted to a jury. You say that we are the jury in this matter. Maybe we are. When you submit a case to a jury, you ask that jury, do you find from a preponderance of the evidence—which is defined to mean the greater weight and degree of credible testimony, that a fact did happen or did not happen. That is what a preponderance of the evidence is, the greater weight of degree of credible testimony.

If there is a doctor in this committee hearing room, I think he or she will tell you that when a question is asked of them do you find, based on a—not what is an absolute fact—but based on the degree of a reasonable medical probability that a fact did or did not happen—going to H.R. 1961 where this resolution says that something may be the result of exposure. On page 3, line 13 that I have already read that such disease "* * * shall be considered to have been incurred * * *" and then you say for us to liberalize the manner in which these claims are disposed, how do you square all of that with what the legal proposition might be when and if this matter is presented to this jury?

Are you asking us to deviate from what is the greater weight and degree of credible testimony and the preponderance of evidence in making a finding, when you testify that the division in the scientific community has continued?

We want to be fair to everybody. We want to be fair to the U.S. Government, too. But can we say with that by a preponderance of the evidence which is the greater weight and degree of credible testimony, that, as of today, we have enough evidence to go to that jury to make a final determination that might affect the lives of millions and millions of people and affect them in a way that is going to cause a drain—and that is not the proper word—but is going to be a tremendous cost factor on the U.S. Government. That is secondary to helping people who need to be and should be helped.

But just taking your testimony alone and the language in this bill, do we have the testimony today, serving as a jury that says we can go out and ask that jury to return a finding of negligence?

Mr. CURRIEO. Not today, you don't. I hope that after the hearings are completed, we will have that evidence.

I would like to draw attention to the remark in my testimony involving Times Beach, Mo. Evidently, the Environmental Protection Agency thought it was necessary to pay millions of dollars to a community because of dioxin poisoning. This is all we are talking about. If there is a correlation, then we must also look at Vietnam veterans. They were also exposed—those who were exposed to agent orange were exposed to dioxin poisoning. If it doesn't affect the Vietnam veterans, it should not affect Times Beach, Mo. We threw away a lot of money in Times Beach, Mo., if that is the case.

Mr. HALL. Do we know that the same factual situation exists in Missouri as existed on Cam Ranh Bay and those other areas, perimeters to those camps?

Mr. CURRIEO. It is very possible.

Mr. HALL. You say it is very possible. Let me interrupt a moment.

You say it is very possible. But if there is still a division in the scientific community—and I am not trying to be the devil's advocate. I am just talking to you now as one veteran to another. I am more concerned about these veterans than I am the other aspects of this thing.

But in order to be fair, are we ready at this point to enact a measure that something may be the result of exposure. Even though we say it may be the result of exposure, on the next page, we are saying that, ". . . such disease shall be considered to have been incurred, notwithstanding that there is no record of evidence of such disease during that period of service."

How can you square that language together and ask us to make a definitive finding on causation.

Mr. CURRIEO. We hope that the hearings will produce scientific evidence, medical evidence, and what other evidence is needed for this committee and for the Congress to make a decision. This is why we are happy that the hearings are being held. This is all we are saying.

We know that the evidence is not here today and that we cannot today ask you to make that decision. Whether the language of the bill will have to be changed, I don't know. But the point is that, as the hearings progress, we hope that scientific evidence and medical evidence is presented and in sufficient quantity for the Members of Congress to make a decision. That is all we are asking. We are just happy that it is being done.

Mr. HALL. Thank you.

I yield back the balance of my time.

Mr. APPLEGATE. The gentleman from South Dakota, Mr. Daschle.

Mr. DASCHLE. Thank you, Mr. Chairman.

I would like to touch on a couple of things Mr. Hall brought up because I have the same interests.

His concern about "may" and "shall," of course, is almost exactly the language used in current law for all presumptive disabilities. So that is not unlike any other 40 presumptive disabilities that we have already passed.

Mr. Hall also asked about the relationship between dioxin exposure in Vietnam and that in Times Beach. Dioxin exposure in Vietnam was 20 times what it was in Times Beach, so we have 20 times the difference.

Then in regard to whether or not there is ample evidence, I think Mr. Walters did the committee a real service in that one particular area, and that is that he addressed the question by stating, "The medical community has reached a consensus that porphyria cutanea tarda, PCT, a liver disorder, can be triggered by dioxin exposure" A relationship also exists between exposure to dioxins and chloracne, a skin disorder that appears to be within a relatively short period of time after exposure. So, obviously, the VA has already reached the conclusion on two of these three diseases.

I want to commend you, Commander Currieo, for your tremendous leadership in what I consider to be one of the most effective organizations on Capitol Hill in relation to the concerns of veterans. You have done an excellent job in presenting the case, in my opinion.

I have a couple of questions that go beyond perhaps the statement itself. What has been the history of the VFW with regard to the other 40 presumptive disabilities? In your organization have you ever seen any administrative problems, any problems with the veterans themselves, in bringing about administrative compensation for these presumptive disabilities?

Mr. CURRIEO. No.

Mr. DASCHLE. Let me ask you a second question. The Ranch Hand study has, of course, been commended and disputed both by the scientific community. Setting aside the scientific elements of the Ranch Hand study, and given the fact that we are talking here about those who were involved with the dispensation of agent orange, from the VFW's point of view, can you draw any distinctions between those who may have been on the ground and those who were in the air?

Mr. CURRIEO. Those who I have talked to—and I have met those who were involved with the Ranch Hand program in Vietnam, as well as those who were in the infantry on the ground—and in my judgment, from those who I talked to and spoke to concerning this issue, those who were on the ground who were exposed, many of them said that they lived days soaked with the defoliant. Those who were used in the Ranch Hand program itself were, of course, exposed, and many times they had an opportunity to wash, to change clothes, and to decontaminate themselves, where those on the ground did not have that opportunity. So this is the difference between those two elements, in my judgment.

Mr. DASCHLE. I appreciate your comments, Commander Currieo.

To me, it has always been kind of like using a hammer. If you are the guy behind the hammer, the chances of getting hurt are a lot less than if you are the guy right in front of it about to get hit. I think that analogy has been lost at times.

Let me ask you about the cardiovascular problems that were studied by the medical followup agency of the National Academy of Sciences. Apparently, on the basis of one study, the VA came to the conclusion that there is a significantly higher risk of dying from diseases of cardiovascular problems and, as a result, have issued a regulation to award service connection for heart disease that develop in veterans with amputations.

They didn't worry about causal relationships in this case. They didn't worry about the specific origination of the problem. They just saw a higher incidence and, on the basis of that, paid compensation. Do you not see a similar kind of a situation here? Isn't there an analogy to be drawn?

Mr. CURRIEO. I believe there is. I firmly believe there is.

Mr. DASCHLE. I, again, applaud your leadership and the kind of attention that this organization has given this issue. Vietnam veterans around the country have taken great pride in knowing that organizations of your reputation, your credibility, and your leadership has brought about, additional action and consideration of this issue here in Congress. We thank you.

Mr. CURRIEO. We thank you, Mr. Daschle.

Also, we again want to express our sincere thanks to all of the members of the committee who have agreed to hear testimony on this important issue. I believe it is finally the giant step forward for the Vietnam veterans.

Mr. APPLEGATE. Thank you, Mr. Daschle.

The gentleman from Mississippi, our distinguished chairman, Mr. Montgomery.

Mr. MONTGOMERY. Thank you, Mr. Chairman.

Thank you, Mr. Commander, Mr. Holt, and Mr. Mayo for being here today.

I have several questions. You mentioned in one of your statements that this is the beginning. Does this mean that the VFW will come back and make recommendations for other disabilities to be presumptive findings, or are you satisfied with this bill and the sunset provision? It is very important to this committee. If you are coming back, we ought to know it.

Mr. CURRIEO. We will have to wait to see what happens in the scientific community. I think that if there is other evidence relating other diseases to dioxin poisoning, I believe it must be liberalized, must be extended, and new legislation must be introduced.

Mr. MONTGOMERY. That is kind of our problem right now, my problem as chairman and also the members of this committee. We get just so much money—I have talked to Mr. Holt about this before. How do we determine our priorities, as far as funding is concerned, and where do we take the money from in the other accounts? This is a problem that I have as chairman of the committee, to determine the type of priorities for the service-connected veteran needing hospital care, or other veterans eligible for a pension. If we move too quickly on this legislation, then we might have to take money away from other accounts.

I will admit this is not an expensive bill and, if it will do the job, that is what we want. And then if we find out from some of these studies that there were Americans affected by agent orange, certainly they are entitled to compensation. But you can see my problem that, if this is just the beginning, then we will have to be very careful how we handle things.

Mr. CURRIEO. Our organization will be reasonable. I am sure everyone concerned will be reasonable in recommending legislation for disability compensation. If there is just evidence that other disabilities should be compensated for, I am sure that the Government will want to see that those veterans are well taken care of. If there is not evidence, then, of course, we will not be asking for more legislation.

Mr. MONTGOMERY. I have another question. Do you think presumptives should be open ended or should there be a reasonable relationship between the period of service from the time when the disability is first noticed?

Mr. CURRIEO. That is a very difficult question to answer. It is going to almost have to—I wouldn't say it should be open ended for ever, but there has got to be a reasonable timeframe in there—we can tell the veteran, "Well, a year after you return from Vietnam, if you haven't come down with cancer or one of the disabilities, then we cannot compensate you." It may take longer. Dioxin poisoning may affect individuals in different manners, just like smoking may affect people in different manners. And it may not affect everyone on the same fashion. So it may take longer for these signs to show up on certain individuals as opposed to others.

Mr. MONTGOMERY. What you said is a reasonable relationship that might go on forever, as far as presumptive is concerned.

Mr. CURRIEO. A good example probably would be in the case of the atomic veterans, those who were exposed to radiation in the atomic tests. Many of those, after 20-some years, are coming forward with problems, and their opportunity to file for disability was cut off very early and they may be really hurting.

Mr. MONTGOMERY. Mr. Commander, a number of your comments have come from the Swedish studies; is that correct?

Mr. CURRIEO. The test results from the Swedish study.

Mr. MONTGOMERY. What are the studies that the VFW has been involved in?

Mr. CURRIEO. We haven't been involved in any studies. We have gathered evidence from the studies, and they are part of the testimony.

Mr. MONTGOMERY. What other studies have you gathered evidence from?

Mr. CURRIEO. From the Swedish studies that were submitted as part of our testimony earlier.

Mr. MONTGOMERY. This is one of the problems I am having. That is why I certainly hope I can keep openminded on this situation. We went to Australia in January, and the Australians were very strong that there was an effect caused by agent orange, and then they did do two detailed studies, and the Australians, in effect, the leadership reversed themselves—this worries me also—they felt like that there were more important issues that money should be spent on for the Vietnam veteran, and that agent orange was not a priority, and that they had psychological problems that needed to be dealt with. There were quick-tempered Australians that were caused from something in the Vietnam war, and they wanted to concentrate more on these other problems that the Vietnam veteran was having in Australia and, in effect, they kind of backed off from agent orange.

Mr. CURRIEO. Also, the Australians, I believe their veterans compensation laws, there is a general presumptive on all disabilities. Those who I have spoke to in Australia who are involved in veterans organizations there indicated that all their disabilities are under a presumptive law.

Mr. MONTGOMERY. I am not sure that is exactly correct. They are paying a number which was ruled by the Federal courts who have the final priority, which is not in our claims system of the veterans. But I was told they are going to move ahead and maybe change some of those presumptive findings.

You are correct. They are paying from 75 to 80 percent of all the claims. Anyone who smoked a cigarette in World War II that was given to them in their food packets by the Australian Government is eligible for claims on heart disease. So they have a problem. What you said is correct.

My time is up. Thank you, Mr. Chairman.

Mr. APPLEGATE. Thank you, Mr. Chairman.

Mr. DASCHLE. Mr. Chairman?

Mr. APPLEGATE. Mr. Daschle.

Mr. DASCHLE. I would like to ask unanimous consent that our chairman have an additional minute. I would like to simply ask an additional question for clarification, if I could.

In regard to the Australian studies, it is my understanding that the first study which has been completed only deals with birth defects.

The morbidity and mortality studies on actual Australian veterans are not yet complete. Is that your understanding also?

Mr. MONTGOMERY. The gentleman could be correct. The study might not be complete. But we met with the officials in Australia, and we were searching for information on agent orange, and the report we wrote—there were 11 members on the committee—we got the impression that it was, in effect, finalized and that they accepted the Senate report for the Australian Government. But there were other area problems more than agent orange.

But in fairness to the gentleman, the Australians did not have as much exposure to agent orange as the American servicemen did.

Mr. DASCHLE. I thank the chairman. That was additional point I wanted to make.

Mr. APPLEGATE. Thank you.

Thank you, Mr. Currieo, Mr. Holt, and Mr. Mayo, for appearing before us.

Mr. CURRIEO. Thank you very much for allowing us to be here this morning.

Mr MONTGOMERY. Mr. Chairman, our colleague Mr. Bonior, during his testimony, mentioned that the subcommittee should subpena certain Dow Chemical records and made special mention of the Midland Dow Co. wives birth defects study.

I have a copy of that study and at this time I would like to place it in the record.

[The material referred to follows:]

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# SURVEY OF REPRODUCTIVE EVENTS OF WIVES OF EMPLOYEES EXPOSED TO CHLORINATED DIOXINS

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**J. C. Townsend, K. M. Bodner, P. F. D. VanPeenen, R. D. Olson, and R. R. Cook (Dow Chemical U.S.A., Health and Environmental Sciences, Midland, MI 48640). Survey of reproductive events of wives of employees exposed to chlorinated dioxins. *Am J Epidemiol* 1982;115:695-713**

**To determine whether paternal exposure to 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) or other polychlorinated dioxins might be associated with adverse pregnancy outcomes, an interviewer-administered questionnaire survey was conducted among wives of Dow Michigan Division employees in the Midland, Michigan, area who had been potentially exposed to dioxins. A control group consisted of wives of employees who had no dioxins exposure and whose hire dates were comparable to those of the men in the exposed group. A total of 737 conceptions, which resulted in 637 live births and 100 stillbirths and spontaneous abortions, were identified as having paternal exposure; 2031 conceptions, resulting in 1785 live births and 246 stillbirths and spontaneous abortions, were identified as having no paternal exposure to any isomer of dioxin. Odds ratios were calculated for dependent variables consisting of spontaneous abortions, stillbirths, infant deaths and several categories of congenital malformations. Trend analysis was performed for duration-of-paternal-exposure of 12 months or less, or more than 12 months. Overall, no statistically significant associations were found between any exposure and pregnancy outcome, either before or after stratification by pertinent sets of up to nine covariables.**

**abortion; congenital defects; dioxins; questionnaires; stillbirth; tetrachlorodibenzodioxin**

This paper describes the results of an interviewer-administered questionnaire survey conducted among women whose husbands were employees of the Midland, Michigan Division of Dow Chemical U.S.A. The purpose of the survey was to compare the proportion of adverse outcomes between pregnancies associated with potential paternal exposures to 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD), or other polychlorinated dioxins, and pregnancies with no known exposures to these chemicals.

Polychlorinated dibenzodioxins, hereaf-

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Abbreviations: FORTRAN, Formula translator, a computer programming language; ICD-8, International Classification of Diseases, Eighth Revision; PL1, Program Language 1, a computer programming language; SAS, Statistical Analysis System, a computer software system; TCDD, 2,3,7,8-tetrachlorodibenzo-p-dioxin; 2,4,5-T, 2,4,5-trichlorophenoxyacetic acid.

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ter referred to as dioxins, may be formed during combustion of carbonaceous fuels or during production of chlorinated phenols (1). Of all the isomers of dioxins, TCDD, a low level contaminant of 2,4,5-trichlorophenol and its byproducts, is considered the most toxic, with an acute single oral $LD_{50}$ in the low $\mu$g/kg range for some laboratory animals (2).

Numerous bacterial or plant test systems have been used to evaluate potential mutagenicity of the dioxins. Hussain et al. (3) found evidence of frameshift mutations with *Salmonella typhimurium*, strain TA 1532, but others have reported doubtful mutagenicity for strains TA 1531 and TA 1534, and non-mutagenicity for strains G-4 and TA 1530 (4).

Mammalian *in vivo* mutagenesis tests have also been performed. Green and Moreland (5) concluded TCDD had no potential for producing chromosomal aberrations in the bone marrow of rats; however, in a subsequent study at higher levels, Green et al. (6) observed an increase which they regarded as weakly positive. Khera and Ruddick (7) exposed male rats to 4, 8 or 12 $\mu$g/kg/day of TCDD for seven consecutive days and then conducted sequential mating trials. Though some levels of the compound caused dose-related toxicity and mortality, there were no dominant lethal mutations noted during the post-treatment period.

A three-generation reproduction study of rats given TCDD, including crossmating with younger untreated animals, indicated adverse reproductive events associated with maternal exposures, but not paternal exposures (8). The latter was true even at the highest dose level, 0.1 $\mu$g TCDD/kg/day (8). Very recently, experiments involving daily oral administration of up to 2.4 $\mu$g/kg TCDD to male mice were reported to have no effect on reproduction or fertility (9).

There have been a series of observational studies of humans related to the mutagenicity or teratogenicity of TCDD or other dioxins. The National Academy of Sciences, commissioned to study the effects of herbicides in South Vietnam, found no conclusive evidence of an association between exposure to herbicides and birth defects in humans (10). Field and Kerr (11) purported a "linear correlation" of neural tube defects and increased use in Australia of the herbicide 2,4,5-trichlorophenoxyacetic acid (2,4,5-T), an herbicide with low levels of TCDD. A similar graph was constructed by Thomas (12) for Hungary where 2,4,5-T usage increased dramatically between 1970 and 1976, but the incidence of stillbirths and congenital malformations remained stable or decreased.

There have been claims of human dioxin-related congenital malformations among offspring of United States and Vietnamese veterans of the Vietnam conflict who were exposed to aerial herbicides spraying (13, 14) and of increased spontaneous abortions among women residing in or near areas of Oregon sprayed with 2,4,5-T (15, 16). Neither of these allegations has been substantiated epidemiologically and the Oregon abortion data, when subjected to critical review, were found not to support the conclusion of an association with herbicide usage (17–19).

Surveillance data on humans have been available following proven contamination of a fairly extensive area with TCDD after a pressure vessel release at a trichlorophenol plant in Seveso, Italy in 1976 (20). Continued monitoring of the population in this area has shown no correlation between spontaneous abortion rates and exposure calculated on the basis of proximity to the factory (21). Although there have been fluctuations in reported congenital malformation rates and spontaneous abortion rates in Seveso during 1976 to 1977, these were reportedly below the Italian national average and there was no pattern consistent with proximity to the site of the release (21). Negative results of cytogenetic evaluations of maternal peripheral blood cells and fetal tissues from TCDD-exposed humans at Seveso have been reported (22–24). The actual degree of exposure to TCDD of husbands and wives in Seveso is unknown, but was sufficient to cause chloracne in 164 children and 23 adults (20).

Dow Chemical U.S.A. has conducted a series of epidemiologic studies of employees potentially exposed to dioxins (25, 26). This survey of reproductive outcomes associated with paternal exposures to dioxins was part of the surveillance program.

## MATERIALS AND METHODS

### *Source of exposed and control populations*

Identification of wives whose husbands had been potentially exposed to dioxins was accomplished via company records. Using annual census lists maintained by the Personnel Department of The Dow Chemical Company, Michigan Division, it was possible to identify employees who had worked in any one of seven buildings associated with chlorophenol processes and hence who were potentially exposed to dioxins. Individual work histories were examined and an employee was considered exposed if he had been assigned for at least one month to specific jobs in the chlorophenol processes between January 1939 and December 1975. By these criteria, a total of 930 male employees with potential dioxins exposures were identified.

An equal number of controls was selected from male employees who, according to work histories, had worked for the company for one month or more, had no job assignments in the potential exposure areas, and closely matched exposed employees by date of hire. Tracing of exposed and control employees was by company records for active employees and retirees receiving benefits. Follow-up of those who had left employment before January 1, 1976, was attempted by submitting names and birth dates to the Michigan Drivers' License Bureau, by searching local telephone directories, by contacting relatives mentioned on work histories, and by submission of names and Social Security numbers to the Social Security Administration. Addresses were thus obtained on all but 69 (3.7 per cent) of the 1860 men. Follow-up was limited to those who lived in Midland or adjacent counties (table 1). Of 930 men in the exposed group, 586 had wives potentially available for interview and 370 agreed to participate. Among the control group, 559 wives were potentially available and 345 were interviewed.

### *Estimation of exposures*

One way dioxins are produced is as byproducts of the manufacture of chlorophenols. Under alkaline conditions, at elevated temperatures and high pressures, bimolecular condensation of chlorophenates may occur. Although theoretically 75 possible polychlorinated dioxins could be formed by condensation of chlorophenols, symmetrically substituted isomers with even numbers of chlorine atoms, including TCDD, were more likely to be formed in the Dow manufacturing processes. Potential dioxins exposures at the Michigan plant existed mainly in the areas which produced or used 2,4,5-trichlorophenol and pentachlorophenol.

Production of pentachlorophenol started in 1939 and 2,4,5-trichlorophenol production started in 1946. Pentachlorophenol was produced by reacting phenol with chlorine, followed by distillation, further chlorination, or treatment with sodium hydroxide, depending on the desired end product. Dioxins associated with these reactions were mostly the higher isomers (hexa-, hepta- and octa-chlorodibenzo-p-dioxins). In 2,4,5-trichlorophenol production, tetrachlorobenzine was hydrolyzed with sodium hydroxide to produce sodium trichlorophenate; TCDD was formed as a byproduct in this reaction and was con-

TABLE 1

*Selection and study participation frequencies among families with husbands never exposed or potentially exposed to dioxins during production operations at the Dow Chemical Company Michigan Division Site, Midland, Michigan, 1939–1975*

| Status | Exposed group | Unexposed group |
|---|---|---|
| Male employees identified as being unexposed or potentially exposed to dioxins | 930 | 930 |
| Employee deceased | 125 | 119* |
| Employee widower or single | 97 | 84 |
| Family residing outside area | 95 | 126 |
| No address available (presumed living) | 27 | 42 |
| Wife eligible for interview | 586 | 559 |
| No response | 18 | 19 |
| Refused | 198 | 194 |
| Interviewed | 370 | 345* |

* It was discovered during the interviews that one woman was widowed, and two others thought to have unexposed husbands were misclassified. Interviews from the two latter were not used, so only 345 of the control interviews were analyzed.

centrated mainly within a caustic insoluble oil layer. The sodium trichlorophenate then was acidified and the resulting 2,4,5-trichlorophenol distilled. In late 1963, a change was made in this process and shortly thereafter a number of employees contracted chloracne. Operations were then modified to minimize exposures. In 1966, 2,4,5-trichlorophenol production was transferred to a new building where new equipment and controls, including automated oil layer dumping, greatly reduced the potential for exposures.

Estimation of exposures was made by an industrial hygienist who was familiar with the processes described above. Based on his experience, information provided by production personnel and surface contamination data, the industrial hygienist estimated exposure potential of jobs within each building in the 2,4,5-trichlorophenol or pentachlorophenol processes. Surface contamination data consisted of wipe tests analyzed by rabbit ear bioassay prior to 1964 and by gas chromatography and mass spectroscopy since 1964. It was presumed that most exposures would have been by skin contact.

Each job was assigned an exposure potential on a scale of low to high for TCDD, for all other dioxins, or for mixed exposures. Jobs involving drying, grinding and packaging of pentachlorophenol and its salt were considered to have had exposure to dioxins other than TCDD. Jobs categorized as low TCDD exposure included 2,4,5-trichlorophenol packers and most 2,4,5-trichlorophenol production operators. The category of moderate TCDD exposures was assigned to mechanics and janitors and to certain operators in 2,4,5-trichlorophenol production and to still operators in the 2,4,5-trichlorophenol distillation area. High potential TCDD exposure was assigned to all jobs involved in the 2,4,5-trichlorophenol process area in late 1963 through 1964, which was when the cases of chloracne occurred (25, 26).

### *Collection and analysis of data*

After cooperation of the local labor union had been obtained, a letter explaining the purpose of the study and requesting a personal interview was mailed to the residence of each living exposed and control population member residing in Midland or adjacent counties. Appointments were made with those women willing to be interviewed. If an employee was a widower or single, or if the response indicated the wife did not wish to be interviewed, no further action was taken. However, if there was no response after several days, a phone call was made to re-explain the intent of the study and to encourage participation. Arrangements were made with those who agreed to be interviewed. No deliberate attempt was made to determine why women did not wish to be interviewed, but unsolicited reasons included divorce, death of spouse, or no pregnancies.

Interviews were conducted in the home by one of the three trained nurse-interviewers. To minimize driving time, appointments were arranged by geographic clusters, rather than by assignment to exposed or control group. Interviewers did not know whether they were interviewing wives of exposed or control employees. At the time of the interview, the intent of the study was re-explained; each woman was advised regarding confidentiality of the information and was asked to sign an informed consent. The interview process normally took less than one-half hour. A five-page questionnaire was used, and answers were recorded in writing on a separate form for each interviewee. Questions were designed to elicit demographic data, marital and family histories, and information considered relevant to pregnancy outcomes. Responses concerning individual pregnancies were used to define and categorize the covariables described below. In addition to questions regarding illnesses associated with pregnancy and the puerperium, there were open-ended questions about unusual events during or immediately after each pregnancy.

Answers were coded and computerized. Responses dealing with health defects, congenital malformations and infant deaths were coded by the Eighth Revision of the *International Classification of Diseases* (ICD-8) (27). Each positive response regarding congenital malformations was coded if the mother stated the condition was diagnosed prior to one year after birth or if the mother was unclear as to time of diagnosis. Conditions identified after the first birthday were reviewed by two physicians. If either physician, blinded as to mother's group, determined the defect could be diagnosed during the first year of life, the condition was so coded. Since these procedures were weighted toward identifying abnormalities, the resulting data could not be meaningfully compared to state or national statistics.

Keypunched data were stored and analyzed on an IBM 370/158 system, with FORTRAN, PL1 and SAS software. For statistical analyses, continuous variables were transformed to categorical; the transformed or generated variables used in analyses are described below in accordance with whether they were considered dependent variables, independent variables or covariables.

Although some complementary analyses were performed using the mother as the observational unit, for the main purposes of this study, the individual conceptus or product of conception was the observational unit, taken as independent of other conceptuses of the same pregnancy or other pregnancies of the same or different women. Effect of exposure to dioxins was conservatively considered as an irreparable event; that is, any exposure of a male employee any time prior to the estimated date of each conception attributed to him was investigated as an independent variable with potential effects on the pregnancy outcome. Conception dates were estimated from the length of pregnancy and pregnancy outcome dates reported in the questionnaire. In many cases, when the date of conception preceded father's first exposure, the conceptuses were assigned to a no exposure category (control) even though the father was in the dioxins group. Two new files were thereby created, one containing only "dioxins" conceptuses and one "non-dioxins" conceptuses; these files were used for subsequent analyses rather than those of the original exposed and non-exposed employees.

### *Variables*

Dependent (outcome) variables were based on the three possible mutually exclusive pregnancy outcomes of live births, miscarriages, and stillbirths. For this study, induced abortions were omitted as outcomes, although they were considered in assigning gravidity scores to subsequent pregnancies. A miscarriage or spontaneous abortion was defined as a non-induced conceptus death with reported length of pregnancy of 20 weeks or less. A live birth was defined as the complete expulsion or extraction of a fetus which breathed or showed other evidence of life. A stillbirth was a conceptus which terminated as other than a miscarriage or a live birth.

A favorable pregnancy outcome was defined as a live birth where the infant survived its first birthday, and was not reported to display a congenital malformation as defined below. Miscarriages, stillbirths, infant deaths, and live births with congenital malformations were considered as unfavorable or adverse outcomes. Infant deaths were live births where the infant died before its first birthday.

Birth defects, which were not necessarily mutually exclusive of infant deaths, were coded only among live births, and were analyzed as either health defects, congenital malformations, or as indicator malformations. A health defect was defined as a live birth reported by the mother to have a health problem of any nature. A congenital malformation was a report by the mother of one or more defects which could be coded to the 740–759.9 range of ICD-8. An indicator malformation was a report by the mother of one or more defects of a set of seven of the following defects (preceded by the ICD-8 code): 741, Spina bifida; 742, Congenital hydrocephalus; 749.0, Cleft palate; 749.1, Cleft lip; 749.2, Cleft palate with cleft lip; 755.0, Polydactyly; 755.1, Syndactyly; and 759.3, Down's syndrome. Thus, indicator malformations were a subset of congenital malformations which, in turn, constituted a subset of health defects. Since mothers and not health professionals were providing the data, it was felt initially that the readily recognizable indicator malformations would provide the most valid outcome measure of the three above. Live births with health defects not codeable as congenital malformations were included as favorable pregnancy outcomes if the infant survived its first birthday.

Independent variables consisted of four exposure categories derived from the industrial hygienist's estimates: TCDD Only, at any level; Any Dioxins (TCDD and others) at any level; Moderate-to-High levels of TCDD only; and, Other Dioxins only (no TCDD). The first two were further broken down into two duration of exposure subcategories: 12 months or less, and more than 12 months. With the break point of 12 cumulative months exposure to dioxins, each subcategory included about one-half of the category-specific conceptuses.

If exposure in a job preceded the calculated date of a conception, the cumulative duration of each type of exposure in months prior to conception was calculated. Conceptuses were considered exposed to TCDD alone only if the father experienced no non-TCDD dioxins exposures before the conception date; those exposed only to non-TCDD dioxins were similarly identified. Conceptuses of dioxins workers were transferred to the control group if there was no paternal exposure to any dioxins prior to conception. Conceptuses of women reporting a marriage date to an exposed husband more than 150 days after the estimated date of conception were also assigned to the control group, since the questionnaire did not ask which conceptions were fathered by the current husband. Conceptions in previous marriages were also assigned to the control group.

For those analyses using the mother as the observational unit, women were allocated into one of four groups: 1) wives of men never exposed, 2) wives whose last conception occurred before their husband's first exposure to dioxins, 3) wives with some conceptions occurring before and some after their husband's first exposure, and 4) wives with all conceptions occurring after their husband's first exposure. The only covariable used in the analyses based on mothers was the total number of conceptions.

Covariables for analyses on conceptus as the observational unit consisted of nine potential confounding variables or effect modifiers (28, 29) which were abstracted from questionnaires. Each covariable, a recent literature reference describing its role, and a description are as follows:

*Age* (30). Mother's age in years at time of conception was computed and then categorized as <18, 18–30, >30 years.

*Birth control method* (31). Use of contraceptives was determined as of time of conception, and included four possible alternatives: none, pills (of any kind), intrauterine device, or any other method.

*Complications during labor and delivery* (32). A large proportion of women reported complications or illnesses after onset of labor, ranging from toxemia to premature rupture of membranes. Interviewers could record two problems with labor and delivery for each conceptus but, because of the diversity of responses, they were treated as a single all-or-nothing measure, i.e., any complication reported for a given conception was taken as a positive response. This variable was only considered in statistical analyses of stillbirths and live births.

*Conditions during pregnancy* (33). The questionnaire permitted a maximum of three maternal medical conditions to be reported as having occurred during each pregnancy. As with complications during labor and delivery, above, a wide variety of responses were elicited, ranging from infectious diseases during pregnancy to diabetes; many could have been causally related to pregnancy outcome. Responses were reduced to a single binary variable, positive (any condition) or negative (no condition reported for any of the questions).

*Medications or treatment during pregnancy* (34). There were two questionnaire items regarding treatments during pregnancy. As above, responses to these items were transformed to a binary variable. Thus, reported treatments such as x-ray and minor medications were given equal weight as a positive response.

*Alcohol consumption* (35). The questionnaire solicited quantitative information about alcohol use during each pregnancy. Responses were divided into two categories: consumption of six or fewer drinks, or more than six drinks per week.

*Gravidity* (36). Three levels of responses were coded: first pregnancy, second or third pregnancy, or fourth or higher pregnancy. Conceptuses of the same pregnancy (twins) were assigned the same value for this variable.

*High risk job* (37). When women were questioned as to their work activities during pregnancy, some named jobs which might have presented a risk to the pregnancy. Responses were reduced to a binary term (risk, no risk) with risk including the following occupations: operating room technician, x-ray technician, bacteriologist, chemical production worker, cosmetologist, and dental assistant.

*Smoking during pregnancy* (38). Interviewers asked the numbers of cigarettes smoked daily during each pregnancy but, for analyses, respondents were grouped as nonsmokers (did not smoke at all during a particular pregnancy) or smokers (smoked at any level during pregnancy).

### *Statistical methods*

For analyses based on the mother as the observational unit, the four groups of mothers were first stratified by their number of conceptions (1,2, . . . 8). The computer program PARCAT (39) was used to obtain various Mantel-Haenszel statistics based on the number of women in each stratum reporting 1,2, . . . 8 pregnancy outcomes of interest (table 2).

The remaining statistical analyses attempted to determine whether there were differences in the distribution of values of outcome variables between the exposed conceptuses and nonexposed conceptuses, after adjusting by stratification for the influences of pertinent covariables. The analyses also attempted to examine, for the independent variables of exposure to TCDD Only or to Any Dioxins, whether there were trends of increasing adverse pregnancy outcomes with increasing durations of exposure.

For 2 × 2 crude tests of exposure-outcome association, as well as for examination of stratum(covariable)-specific associations, chi-square statistics were generated. Where chi-square was not appropriate due to table sizes, Fisher's exact test was employed (40). Tests for trend in 2 × 3 tables employed chi-square one degree-of-freedom statistics with linear weights (40). The corresponding tests on stratified sets of tables used Mantel-Haenszel chi-square statistics (41) and extended Mantel-Haenszel statistics (42). Programs to generate all of the above were written in SAS and SAS PROC MATRIX (43).

A FORTRAN program by Thomas (44) was used for calculation of odds ratios and related statistics to supplement chi-square tests.

For stratified analyses, Thomas's program computed asymptotic maximum likelihood odds ratios and confidence limits using a modification of the Extended Cornfield Method (45). An asymptotic test of the normal deviate, calculated by Mantel-Haenszel estimates of expected value and variance, was used as the one-tailed main effects test of departure of the odds ratio from unity. An asymptotic chi-square test for homogeneity (departure of the stratum-specific odds ratios from the overall stratified estimate of the odds ratio) was also performed. The latter two tests yielded *p* values only.

For stratum-specific calculations, where table sizes were <500, Thomas's program yielded a maximum likelihood estimate of the odds ratio, 95 per cent confidence limits, and an exact test of the stratum odds ratio against unity, giving a one-tailed *p* value. Where table sizes were large, as was the case for crude contingency tables, asymptotic tests and estimates were produced instead of exact estimates.

The effect of covariables on the association between exposure and pregnancy outcome was based on their independent action, their interaction with other independent or covariables, and/or their distribution at the combinations of levels of independent variables and covariables. It was not expected that a particular covariable would have the same effect (if any) on one contingency table of exposure versus outcome as on another table with different measures of exposure or outcome. Therefore, for each of the 48 exposure-outcome analyses, the set of nine potential covariables was evaluated using a forward-selection algorithm adapted to categorical data by Higgins and Koch (46). This algorithm selected a covariable set by assessing the relative strength of association of each candidate covariable with the outcome variable at each level of the covariables already accepted into the study. However, instead of using sets of 2 × 2 tables, as did Higgins and Koch, our procedure used extended ($r \times c$, $r, c > 2$) chi-square, and Cochran-Mantel-Haenszel chi-square measures to select covariables. We omitted the independent exposure variable from the candidate set of variables until the selection was complete instead of allowing it into the model first. The selection procedure stopped when no further associations of covariable-outcome were found to be significant at the $\alpha = 0.10$ level.

TABLE 2

*Distribution of conception frequencies of wives of the Dow Chemical Company Michigan Division Site employees unexposed and potentially exposed to dioxins during production operations, Midland, Michigan, 1939–1975*

| No. of conceptions | No. of women with conceptions placed relative to husband's first exposure | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | Group 1 | | Group 2 | | Group 3 | | Group 4 | | Total groups 2–4 | |
| | Husband never exposed | | All conceptions pre-exposure | | Some conceptions pre-exposure | | All conceptions post-exposure | | | |
| | No. | (%) | No. | (%) | No. | (%) | No. | (%) | No. | (%) |
| 1 | 22 | ( 7) | 4 | ( 4) | 0 | ( 0) | 13 | (12) | 17 | ( 5) |
| 2 | 60 | (18) | 23 | (23) | 22 | (15) | 22 | (21) | 67 | (19) |
| 3 | 82 | (25) | 32 | (31) | 33 | (22) | 23 | (22) | 88 | (25) |
| 4 | 63 | (19) | 17 | (17) | 24 | (16) | 19 | (18) | 60 | (17) |
| 5 | 56 | (17) | 13 | (13) | 21 | (14) | 13 | (12) | 47 | (13) |
| 6 | 20 | ( 6) | 6 | ( 6) | 13 | ( 9) | 6 | ( 6) | 25 | ( 7) |
| 7 | 14 | ( 4) | 4 | ( 4) | 16 | (11) | 5 | ( 5) | 25 | ( 7) |
| 8 | 6 | ( 2) | 2 | ( 2) | 10 | ( 7) | 1 | ( 1) | 13 | ( 4) |
| 9+ | 9 | ( 3) | 1 | ( 1) | 12 | ( 8) | 4 | ( 4) | 17 | ( 5) |
| | (88 conceptions) | | (11 conceptions) | | (128 conceptions) | | (45 conceptions) | | (184 conceptions) | |
| Total | 332* | | 102 | | 151 | | 106 | | 359* | |
| | (1274 conceptions) | | (370 conceptions) | | (742 conceptions) | | (391 conceptions) | | (1503 conceptions) | |

* Twenty-four women, 13 in Group 1 and 11 in Group 2, 3, or 4 reported no pregnancies at interview.

## RESULTS

As noted previously, interviews were obtained from 370 wives (63 per cent) of the 586 potentially exposed employees eligible for interview, and 345 (62 per cent) of the 559 eligible control employees. Among eligible members of two previously studied cohorts, a "chloracne cohort" (25) and a "2,4,5-T cohort" (26), participation rates in the current study were comparable (67 per cent and 59 per cent, respectively).

The distribution of conception frequencies among wives of unexposed (group 1) and exposed (group 2, 3 or 4) employees is shown in table 2. A total of 1274 conceptions were reported by unexposed women, and 1503 by women in the exposed groups. Eleven unexposed and 13 exposed workers' wives claimed no pregnancies at interviews.

To examine the potential for recall bias, comparisons of frequencies of various adverse pregnancy outcomes between group 1 (nonexposed) and group 2 (all conceptions pre-exposure) women were made. No chi-square statistics so generated were found to approach significance.

As a test for exposure effects using the mother as the observational unit, adverse outcome frequencies of group 1 (nonexposed) and of group 2 (all conceptions pre-exposure) women were compared with those of group 4 (all conceptions post-exposure) women. Again, no comparisons detected any significant differences.

The bulk of the analyses were based on the conceptus as the observational unit. The homogeneity of outcomes of group 1 and group 2 women demonstrated above suggested that conceptions of nonexposed women, and group 2 women, and all conceptions of group 3 women occurring prior to the date of first exposure could be pooled as a control group of conceptions. Two thousand thirty-one conceptions, including 1785 live births, were thus identified as being nonexposed, or "non-dioxins" conceptions. Seven hundred thirty-seven conceptions, including 637 live births, were similarly counted among group 4 women (all conceptions post-exposure) and among post-exposure pregnancies of group 3 women. These were identified as "dioxins-exposed" conceptions for subsequent analyses. Nine induced abortions were deleted from the analyses.

Characteristics of the "dioxins" and "non-dioxins" conceptions and their frequencies are listed in tables 3–5. Table 3 shows frequencies of the coded diagnoses for all congenital malformations or health defects and for infant deaths. The frequency distribution of covariable values for conceptuses in the TCDD Only, in the Any Dioxins, and in the unexposed groups, are listed in table 4. Some differences were obvious; there were proportionately more conceptuses with mothers who were older than 30 years of age, or who were gravida 4+ in the exposed group, while there were proportionately more conceptuses with mothers who were taking medications or who had an illness during pregnancy in the control group. All multiple births were twins, with 35 sets comprising a total of 70 twins. Twins were proportionately equally distributed among dioxins conceptuses (10 sets in 737 or 1.36 per cent) and non-dioxins conceptuses (25 sets in 2031 or 1.23 per cent).

Table 5 shows the frequencies of various adverse pregnancy outcomes or dependent variables for each paternal exposure category, and the rates and ratios per 1000 live births calculated from these numbers. It should be emphasized that data in table 5 are crude and represent frequencies which were not adjusted for the influence of any covariables.

Numbers were very small in the independent variable categories of Moderate-to-High TCDD and of Other Dioxins alone, and in the dependent variable category of Indicator Malformations. These will therefore not be considered further as separate categories in the sections on Re-

TABLE 3

*Frequencies and classification of incidents of infant death and health defects among live births to wives of Dow Chemical Michigan Division employees unexposed and potentially exposed to dioxins during production operations, Midland, Michigan, 1939–1975*

| Classification of death or illness | ICD-8 codes | Frequency |
|---|---|---|
| I. Congenital malformations or health defects* | | |
| All congenital anomalies | 740–750.9 | 130 |
| Spina bifida | 741 | 1 |
| Congenital anomalies of heart | 746 | 12 |
| Cleft palate and cleft lip | 749 | 10 |
| All others | 743–5; 748; 750–9 | 107 |
| Other causes of perinatal morbidity and mortality | 778–9 | 17 |
| Anoxic and hypoxic conditions NEC | 776 | 14 |
| Birth injury and difficult labor | 764; 768; 772 | 12 |
| Benign neoplasms and neoplasms of unspecified nature | 227; 232 | 11 |
| Intestinal obstruction and hernia | 550–1; 560 | 9 |
| All others | 112; 172–3; 254; 275; 343; 373; 389; 427; 429; 482; 511; 520; 564; 572; 607; 685; 692; 738; 771; 775; 781 | 46 |
| II. Infant deaths | | |
| Anoxic and hypoxic conditions NEC | 776 | 10 |
| Other causes of perinatal morbidity and mortality | 777 | 10 |
| Congenital anomalies of heart, and other anomalies | 746; 742; 748; 751; 759 | 5 |
| Conditions of placenta and cord | 770; 771 | 5 |
| Pneumonia | 485–6 | 5 |
| All others | 246; 254; 320; 323; 508; 593; 772; 775; 795; 796 | 13 |

* A single conceptus may have contributed none, one or two codeable diagnoses, so numbers will not add up to total live births.

sults and Discussion, although they were analyzed as such when possible, and the Moderate-to-High-TCDD category was, of course, included in the TCDD-Only category. Table 6 is a summary of the crude and adjusted odds ratios with their 95 per cent confidence limits. Table 6 also lists $p$ values for tests of homogeneity among stratum-specific odds ratios. As shown, no statistically significant differences in proportions of adverse pregnancy outcomes were detected using crude or adjusted odds ratios from 2 × 2 summary tables. Similarly, although not shown, results of the analyses for the other two exposure categories (Other Dioxins alone, and Moderate-to-High TCDD alone) were not statistically significant. All Mantel-Haenszel chi-square values, as well as crude chi-square statistics, were low, having $p$ values greater than 0.10. Crude and adjusted trend analyses based on the length-of-exposure categories described previously were also nonsignificant.

TABLE 4

*Characteristics of pregnancy environments of 2768 conceptions classified as unexposed or potentially exposed to dioxins through paternal work experiences in Dow Chemical Michigan Division production areas, Midland, Michigan from 1939–1975*

| Covariable | Exposure category of conceptus | | | | | |
|---|---|---|---|---|---|---|
| | Any dioxin | | TCDD† only | | Unexposed | |
| | No. | (%) | No. | (%) | No. | (%) |
| I. Mother's age (years) at conception | | | | | | |
| Under 18 | 8 | ( 1) | 4 | ( 1) | 113 | ( 6) |
| 18–30 | 539 | (73) | 322 | (77) | 1675 | (82) |
| Over 30 | 190 | (26) | 92 | (22) | 243 | (12) |
| II. Birth control method used at time of conception | | | | | | |
| None | 658 | (89) | 378 | (90) | 1884 | (93) |
| Pills | 14 | ( 2) | 6 | ( 1) | 12 | ( 1) |
| Intrauterine device | 5 | (<1) | 1 | (<1) | 7 | ( 1) |
| Other | 60 | ( 8) | 33 | ( 8) | 128 | ( 6) |
| III. Complications or illnesses during labor and delivery* | | | | | | |
| None | 456 | (72) | 271 | (74) | 1278 | (72) |
| Any | 181 | (28) | 97 | (26) | 507 | (28) |
| IV. Health problems of mother during pregnancy | | | | | | |
| None | 482 | (65) | 269 | (64) | 1447 | (72) |
| Any | 255 | (35) | 149 | (36) | 584 | (28) |
| V. Mother's medication or treatment during pregnancy | | | | | | |
| None | 473 | (64) | 257 | (61) | 1382 | (68) |
| Any | 264 | (36) | 161 | (39) | 649 | (32) |
| VI. Mother's average weekly alcohol intake during pregnancy | | | | | | |
| Six or fewer drinks | 721 | (98) | 405 | (97) | 2020 | (99) |
| More than six drinks | 16 | ( 2) | 13 | ( 3) | 11 | (<1) |
| VII. Gravidity | | | | | | |
| 1st pregnancy | 96 | (13) | 62 | (15) | 603 | (30) |
| 2nd or 3rd pregnancy | 297 | (40) | 186 | (45) | 890 | (44) |
| 4th or higher pregnancy | 344 | (47) | 170 | (41) | 538 | (26) |
| VIII. Did mother work in high risk activity during pregnancy | | | | | | |
| No | 726 | (99) | 416 | (100) | 2008 | (99) |
| Yes | 11 | (<1) | 2 | (<1) | 23 | ( 1) |
| IX. Mother's smoking pattern during pregnancy | | | | | | |
| Nonsmoker | 528 | (72) | 320 | (77) | 1566 | (77) |
| Smoker | 209 | (28) | 98 | (23) | 465 | (23) |

* Applies only to live births and stillbirths (not to spontaneous abortions).

† TCDD, 2,3,7,8-tetrachlorodibenzo-p-dioxin.

Although none of the overall odds ratios, crude or adjusted, were statistically significant, there was noticeable lack of homogeneity among stratum-specific odds ratios for some of the exposure-outcome associations shown in table 6. This occurred among the outcome categories of Spontaneous Abortions, all Conceptus Deaths, and all Unfavorable Outcomes for the Any Dioxins exposure category, and among the same three for the TCDD-Only category. Consequently, all covariable-specific subtables for these six exposure-outcome combinations were examined for significant associations. Seven such associations were found, five with odds ratios greater than one, and two with odds ratios less than one. Recurrent

Table 5

*Frequencies and rates or ratios (per 1000 live births) of pregnancy outcomes of 2768 conceptions classified as unexposed or exposed to dioxins through paternal work experiences in Dow Chemical Michigan Division production areas, Midland, Michigan, from 1939–1975*

| | Exposed | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| Pregnancy outcome | TCDD† alone | | | | Other dioxins alone | Any dioxin | | | Control | Total |
| | Moderate to high | Any level | | | | ≤1 year | >1 year | Total | | |
| | | ≤1 year | >1 year | Total | | | | | | |
| Conceptus deaths | | | | | | | | | | |
| Miscarriage | 0 | 18 | 25 | 43 | 0 | 36 | 49 | 85 | 213 | 298 |
| (Ratio) | (00.0) | (108.4) | (123.8) | (116.8) | (00.0) | (112.5) | (154.6) | (133.4) | (119.3) | (123.0) |
| Stillbirth | 0 | 3 | 4 | 7 | 0 | 8 | 7 | 15 | 33 | 48 |
| (Ratio) | (00.0) | (18.1) | (19.8) | (19.0) | (00.0) | (25.0) | (22.1) | (23.5) | (18.5) | (19.8) |
| Total | 0 | 21 | 29 | 50 | 0 | 44 | 56 | 100 | 246 | 346 |
| (Ratio) | (00.0) | (126.5) | (143.6) | (135.9) | (00.0) | (137.5) | (176.7) | (157.0) | (137.8) | (142.9) |
| Live births | | | | | | | | | | |
| Health defect | 2 | 18 | 14 | 32 | 2 | 26 | 26 | 52 | 155 | 207 |
| (Rate) | (181.8) | (108.4) | (69.3) | (87.0) | (100.0) | (81.3) | (82.0) | (81.6) | (86.8) | (85.5) |
| Congenital malformation | 0 | 14 | 7 | 21 | 1 | 19 | 11 | 30 | 87 | 117 |
| (Rate) | (00.0) | (84.3) | (34.7) | (57.1) | (50.0) | (59.4) | (34.7) | (47.1) | (48.7) | (48.3) |
| Indicator malformation | 0 | 1 | 1 | 2 | 0 | 1 | 2 | 3 | 10 | 13 |
| (Rate) | (00.0) | (06.0) | (05.0) | (05.4) | (00.0) | (03.1) | (06.3) | (04.7) | (05.6) | (05.4) |
| Infant death | 0 | 4 | 2 | 6 | 1 | 7 | 2 | 9 | 39 | 48 |
| (Rate) | (00.0) | (24.1) | (09.9) | (16.3) | (50.0) | (21.9) | (06.3) | (14.1) | (21.8) | (19.8) |
| Total unfavorable* | 0 | 16 | 8 | 24 | 2 | 24 | 12 | 36 | 120 | 156 |
| Total favorable | 11 | 150 | 194 | 344 | 18 | 296 | 305 | 601 | 1665 | 2266 |
| Total | 11 | 166 | 202 | 368 | 20 | 320 | 317 | 637 | 1785 | 2422 |
| All conceptions | | | | | | | | | | |
| Total unfavorable | 0 | 37 | 37 | 74 | 2 | 68 | 68 | 136 | 366 | 502 |
| (Rate) | (00.0) | (197.9) | (160.2) | (177.0) | (100.0) | (186.8) | (182.3) | (184.5) | (180.2) | (101.4) |
| Total | 11 | 187 | 231 | 418 | 20 | 364 | 373 | 737 | 2031 | 2768 |

* Unfavorable outcomes include only malformations with ICD-8 codes 740–759.9.

† TCDD, 2,3,7,8-tetrachlorodibenzo-p-dioxin.

TABLE 6

*Crude and covariate-adjusted estimates of risk for adverse pregnancy outcomes among 2768 conceptions unexposed or potentially exposed to dioxins through paternal work experiences in Dow Chemical Michigan Division production areas, Midland, Michigan, from 1939–1975*

| Dependent variable | Crude Risk | | Risk Adjusted by Stratification of Covariates | | |
|---|---|---|---|---|---|
| | Odds ratio | (95% confidence limits) | Odds ratio | (95% confidence limits) | *p* (homogeneity of subtables) |
| I. Exposure to any dioxin | | | | | |
| All conceptus deaths | 1.14 | (0.88, 1.47) | 1.03 | (0.78, 1.37) | < 0.01 |
| Stillbirths | 1.27 | (0.66, 2.45) | 1.06 | (0.54, 2.09) | 0.20 |
| Spontaneous abortions | 1.12 | (0.85, 1.47) | 1.03 | (0.77, 1.39) | 0.02 |
| Infant deaths | 0.64 | (0.29, 1.39) | 0.63 | (0.27, 1.39) | 0.61 |
| Health defects | 0.93 | (0.66, 1.31) | 0.85 | (0.60, 1.21) | 0.57 |
| Congenital malformations | 0.96 | (0.62, 1.50) | 0.85 | (0.53, 1.35) | 0.62 |
| All unfavorable outcomes* | 1.03 | (0.82, 1.29) | 0.97 | (0.76, 1.22) | 0.05 |
| II. Exposure to TCDD† only | | | | | |
| All conceptus deaths | 0.99 | (0.70, 1.40) | 1.02 | (0.71, 1.47) | 0.09 |
| Stillbirths | 1.03 | (0.41, 2.45) | 0.97 | (0.38, 2.36) | 0.37 |
| Spontaneous abortions | 0.98 | (0.68, 1.40) | 0.96 | (0.65, 1.42) | 0.06 |
| Infant deaths | 0.74 | (0.28, 1.85) | 0.82 | (0.30, 2.09) | 0.42 |
| Health defects | 1.00 | (0.66, 1.52) | 0.93 | (0.60, 1.43) | 0.60 |
| Congenital malformations | 1.18 | (0.70, 1.97) | 1.08 | (0.63, 1.83) | 0.86 |
| All unfavorable outcomes* | 0.98 | (0.74, 1.30) | 0.98 | (0.72, 1.32) | 0.06 |

* Unfavorable outcomes include only malformations with ICD-8 codes 740–759.9.

† TCDD, 2,3,7,8-tetrachlorodibenzo-p-dioxin.

in the subtables with significantly elevated odds ratios was a group of nine TCDD-exposed conceptions which contributed three spontaneous abortions and only six live births to the analyses. Mothers of these conceptuses characteristically were 18–30 years of age, gravida 4+, were not using birth control methods at conception, reported no illnesses during pregnancy, did not work at high risk jobs, took medications during pregnancy, and had fewer than seven drinks per week. Except for slight differences in the pertinent covariable sets, TCDD-Only exposure is a subset of Any Dioxins exposure, and Spontaneous Abortions is a subset of Conceptus Deaths which, in turn, is a subset of Unfavorable Outcomes. Therefore, the nine conceptions account for much of the significant association in the other covariable-specific subtables. Detailed examination of subtables in the trend analyses gave similar conclusions.

Statistically significantly elevated or decreased odds ratios were not obtained for any other covariable-specific group. In fact, any changes in covariable category selection for the covariable-specific group described above (i.e., changing the age category, lowering gravidity, changing birth control usage to a different method or changing medications use from yes to no) not only eliminated statistical significance but in two cases significantly changed the direction of the association.

## DISCUSSION

As detailed in the section on Results above, there were no statistically significant differences in crude or overall adjusted odds ratios for adverse pregnancy outcomes and any of the exposure categories, nor were there significant trends overall with increased duration of exposure on a crude or adjusted level.

The lack of an association of adverse pregnancy outcomes with differences in paternal exposure to dioxins may have at least five possible explanations: 1) differences did occur but were too small to be detected given the numbers involved and kinds of tests used; 2) differences did occur but could not be detected because of confounding by variables other than those considered; 3) differences did occur but were not detected because of inappropriate selection of exposed and unexposed populations; 4) differences did occur but were not detected because of weaknesses of the methods or errors in conduct of the study; or 5) paternal workplace exposures to dioxins were not associated with increased numbers of adverse pregnancies.

As regards the first possible explanation, ratios of adverse pregnancy outcomes have shown considerable unexplained fluctuations in almost every population which has been studied (47), and such fluctuations may have obscured differences here. From a purely pragmatic viewpoint, we could not have increased the power to detect differences by increasing the size of the exposed population since all employees with potential dioxins exposure were identified at the outset of this study. It might have been possible to increase power by increasing the number of controls (48), but the ratio of control conceptuses to exposed was already almost 3:1 (2031/737), and was even greater for exposure subsets. Despite these caveats, we did calculate the power (one minus beta error) possible at various levels of odds ratios for crude data in our study according to the program of Rothman and Boice (49). Depending upon which outcome was tested (and, hence, what the background or unexposed rates were), at $\alpha = 0.05$, the power to detect a crude odds ratio of 1.5 or greater varied from less than 50 per cent for Stillbirths, Indicator Malformations and Infant Deaths to 80 per cent or greater for all Conceptus Deaths, all Unfavorable Outcomes, and for Spontaneous Abortions. The power to detect odds ratios of two or greater was more than 90 per cent for these last three, whereas it was 70 per cent for Stillbirths, 98 per cent for Health Defects, 93 per cent for Congenital Malformations, 26 per cent for Indicator Malformations, and 61 per cent for Infant Deaths.

Unidentified covariables, if unequally distributed, may have obscured differences in pregnancy outcomes between exposed and control groups, even though we attempted to identify such variables and we chose statistical methods to handle confounding and effect modification. In addition, there is a practical limit to the number of variables which can be included in analyses.

Inappropriate selection of exposed and control populations can be a problem in any epidemiologic study. Lack of specific exposure data, and dilution of exposure groups with individuals who were not actually exposed could be considered weaknesses of any chemical industry epidemiologic studies. However, chemical exposures usually cannot be precisely documented. In the present study, exposed employees were selected on the basis of potential exposure to dioxins, and we attempted to quantify potential exposures as described previously. This quantification was relative because analytical methods sensitive enough to detect levels less than one part per million of dioxins were not available until refinements in gas chromatography were introduced in our analytical laboratories in the 1970's. However, since some employees in the high TCDD group had developed chloracne, there is relatively objective evidence that at least some had experienced fairly high exposures to dioxins. In addition, separation of the exposed group into two levels of duration of potential exposures permitted evaluation of dose-response.

It might be argued that controls selected from the same industry are inappropriate, but it is well known that employed persons differ in important health-related respects from the general population. Since the objective of this study was to identify effects of dioxins exposure rather than effects of other chemical exposures, it seemed reasonable that the control population be selected in exactly the same manner as the exposed.

There are weaknesses inherent in use of questionnaires to acquire epidemiologic data. Candidates for interview who were not available or eligible for interview or who refused to participate might, in fact, be the very persons most at risk. However, the refusal rates for both exposed and control wives were similar (198/568, 35 per cent and 194/540, 36 per cent) and there is no reason to suspect that reasons for noneligibility or refusal were different in the two groups. In addition, participation among the two previously studied cohorts mirrored the overall participation rates.

Bias of interviewers and respondents was possible, although an attempt was made to blind interviewers. To comply with the intent of informed consent, respondents knew the study concerned employees who had dioxins exposure and those who did not, but often the wife did not know whether her husband worked in a potential dioxins-exposed area or, if so, whether this applied to a particular pregnancy.

While statistical tests failed to reject the null hypothesis of no recall bias, it should be recognized that such a bias, if operating, would result in risk estimates erroneously elevated. However, there were no overall risk estimates that were statistically significantly elevated. We tried to avoid interpretation variation and related errors by having the same individual handle all data coding for computer input.

The analytical methods were selected as best suited for the multiple analyses needed for the many dependent and independent variables and covariables. Fitting covariable questionnaire data into categories may not always have been appropriate; for example, some illnesses and some medications could be considered more important than others. In addition, the variable selection procedure for assessing the relative strengths of association between covariables and outcome was not always appropriate because of heterogeneity of component strata. Small table sizes, particularly for subtables, often resulted in numbers insufficient for statistical analyses. Nevertheless, the variable selection process used seemed a reasonable compromise between an all-possible-combinations approach, where every level of every covariable is treated independently, and a complete modeling approach. That the method used was, in fact, capable of detecting differences was demonstrated by identification of a covariable-specific group with odds ratios statistically significantly different from one at the $\alpha = 0.05$ level as reported in the results.

We are uncertain as to why the covariable-specific groups in question should have yielded elevated odds ratios. One possibility was that subcategories within both the dependent and independent variables were not mutually exclusive. Spontaneous Abortions are a subset of Conceptus Deaths which are, in turn, a subset of Unfavorable Outcomes. In addition, the TCDD-Only exposure category is a subset of Any Dioxins exposure. In fact, a large component of the significant associations could be traced to nine conceptuses in the TCDD-Only exposed group with three spontaneous abortions and only six live births mentioned previously. By contrast, there were only one spontaneous abortion and 41 live births among the controls for this covariable-specific group. The relatively high number of abortions (for this study) in these exposed and the unusually low number in the controls accounted for some of the magnitude of the observed odds ratios.

Another possibility was that of the positive results occurring by chance because of the large number of analyses performed on the data. Theoretically, 73,728 2 × 2 tables were possible since there were totals of eight possible dependent variables, four possible independent or exposure variables and nine possible covariables. Six of these covariables had only two possible levels, but two (age and gravidity) had three and one (birth control method) had four. However, the covariable selection procedure reduced this possible number to 3505 and, of these, many tables either would have included no conceptuses at all or none in one of either the dependent or independent category levels. As a result, only 259 tables were actually tested, and odds ratios from only seven were found to be statistically significantly different from unity: five were significantly elevated and two significantly decreased. In the trend analyses, there were 36,864 theoretically possible tables, 2058 remained after covariable selection and 200 tables were actually prepared and tested; of these, six showed a statistically significant trend of increasing odds ratios with increased durations of exposure. As noted below, these positives were based on the same covariable-specific groups which were already positive in the 2 × 2 tables.

Statistically significant odds ratios were all for the dependent variables of Spontaneous Abortions, Conceptus Deaths, or Unfavorable Outcomes except in the case of the two decreased odds ratios which were for Spontaneous Abortions and Infant Deaths. In all cases, the covariable-specific groups of conceptuses were from women between the ages of 18 and 30 years, who did not use birth control methods, drank six or less alcoholic drinks per week and, except for one table, did not have a high risk job during pregnancy.

An alternative explanation to those preceding, of course, is that there was a real exposure effect which, for some reason, was limited to the covariable-specific group described. This is inconsistent with the overall nonpositive results of the study.

The fifth and last explanation offered for our results, that there were no differences in adverse pregnancy outcomes, is consistent with what is known of biological effects of the dioxins. While the experimental literature suggests that maternal exposures to some levels of TCDD may be associated with embryonic and teratogenic events in laboratory animals, the experimental literature also indicates that paternal exposures do not put conceptuses at excessive risk to unfavorable outcomes (7−9). The negative studies of humans exposed to relatively high levels of TCDD in Seveso, Italy, reinforce the laboratory data (20−24).

In conclusion, it is our interpretation that, under the conditions of this study, there were no biologically meaningful associations between adverse pregnancy outcomes and paternal dioxins exposures.

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Mr. MONTGOMERY. Mr. Chairman, we might have some additional questions that I would like to submit to our witnesses for the record.

Mr. APPLEGATE. Without objection, they will be included in the record.

[The information appears on p. 275.]

Mr. APPLEGATE. Next we have Mr. Donald Swartz, national director of veterans' affairs, Veterans of the Vietnam War; Michael Milne, national director, Veterans of the Vietnam War; and Mr. James Roxby, the Pennsylvania State Director of the Veterans of the Vietnam War.

While the gentlemen are coming to the table, we also have with us a very distinguished Congressman, Frank Harrison, from the great State of Pennsylvania. Mr. Harrison.

Mr. HARRISON. Thank you, Mr. Chairman.

I would like to thank the committee and, in particular, you, for allowing me to sit with you this morning. I am not a member of the subcommittee. I am, of course, interested in the subject matter before you in this hearing.

The witnesses who have just come to the table are constituents and friends of mine who have gained considerable insight into this question—Mr. Swartz, through his everyday work as a director of the Governor's Veterans Outreach Center, Mr. Milne and Mr. Roxby through means that I think are personally more painful.

I appreciate the committee taking time to hear them this morning. I hope to participate in a dialog with them when they finish their testimony.

Mr. APPLEGATE. Thank you, Mr. Harrison.

## STATEMENTS OF DONALD J. SWARTZ, NATIONAL DIRECTOR OF VETERANS' AFFAIRS, VETERANS OF THE VIETNAM WAR; MICHAEL MILNE, NATIONAL DIRECTOR, VETERANS OF THE VIETNAM WAR; AND JAMES ROXBY, PENNSYLVANIA STATE DIRECTOR, VETERANS OF THE VIETNAM WAR

Mr. SWARTZ. Good morning, Mr. Chairman.

Mr. Chairman and members of the subcommittee, I am honored to appear before the Subcommittee on Compensation, Pension and Insurance to discuss my views on H.R. 1961.

This bill would amend title 38 of the United States Code, to provide a presumption of service connection for the occurrence of certain diseases related to exposure to herbicides or other environmental hazards or conditions in veterans who served in Southeast Asia during the Vietnam era.

I am a 4-year veteran of the U.S. Navy, of which the last 2 years were spent as a corpsman attached to the Marine Corps; 8 months out of those last 2 years were served in Vietnam.

First of all, I would like to take this opportunity to sincerely thank Mr. Daschle for introducing H.R. 1961 and the 113 cosponsors for having the courage and concern for the Vietnam veteran to tackle such an emotional and controversial issue.

The Centers for Disease Control has rated any level of dioxin above 1 part per billion as posing a serious risk to health. Thousands of Vietnam veterans for years have flocked to the Veterans' Administration complaining of a myriad of problems, yet these complaints have fallen on deaf ears. The Vietnam veteran has been slapped in the face enough. How many more times must we turn the other cheek? We have seen the Environmental Protection Agency issue a ban on the production of 2,4,5-T which produces the dioxin in agent orange. We have seen the entire community of Times Beach, Mo., evacuated at Government expense because of exposure to dioxin. Yet, the Veterans' Administration persists in its hard line, that there is no proof that dioxin is harmful to one's health. How can it be that dioxin is harmful to the people in Times Beach where the levels of dioxin were far less than what we were exposed to, and yet not harmful to the Vietnam veteran? We were not innoculated against this toxin, nor were we informed of the possible adverse health effects while serving in Asia.

On August 27, 1982, we again had to turn the other cheek. The New York Times published an article concerning 47 railroad workers who were exposed to dioxin. They were awarded $58 million in settlement. These workers developed medical problems indentical to those the Vietnam veterans have been complaining about. The amount of dioxin these workers were exposed to was 22 parts per billion—again, far, far less than the levels of dioxin in agent orange used in Southeast Asia.

Rarely a day goes by that one doesn't read something about a community or workers being exposed to dioxin, and the concern that their exposure invokes. Yet, where is the concern for the Vietnam veteran? Just as the Vietnam Veteran Memorial dedication last November was a step in the right direction to finally recognize the sacrifices of the Vietnam veterans, so, too, is H.R. 1961 a step in the right direction—but only a step in what will be a long, long journey.

On page 3 of this bill, it itemizes the conditions that would be granted presumption for service connection, and I definitely concur with this judgment. However, what do we tell those Vietnam veteran who are suffering from other medical conditions known to be induced by exposure to dioxin? Do we once again ask them to turn the other cheek? I implore you to expand this bill to include these other known medical conditions.

Item (2)(A)(IV) of this bill would allow the Administrator of the Veterans' Administration to include other diseases that medical research shows causes health problems. I have serious reservations with this portion of H.R. 1961. In view of the hard-line approach of the Veterans' Administration handling of the agent orange issue, I must say that I view their having final say on what diseases will or will not be included with much skepticism. If one feels this skepticism is unwarranted, then I suggest you read the General Accounting Office report to Congress, dated October 25, 1982, regarding the Veterans' Administration's handling of the agent orange issue.

Therefore, I suggest that the Centers for Disease Control or some other agency evaluate the known literature on dioxin and its adverse health effects and determine what other diseases should be granted service connection, and that the Veterans' Administration be mandated to accept their findings and grant disabilities to those who qualify.

Approximately 106,000 veterans have taken the agent orange examination at the Veterans' Administration hospitals throughout the country. That certainly is a large enough population to draw conclusions about various health problems. If it is obvious that certain trends developed, then why must we wait until 1991, when the Centers for Disease Control epidemiological study will be concluded, for action. Again, if the only common factor is service in Vietnam, then the only conclusion that can be reached is that it must be a service-connected disability due to one's service in Vietnam.

In my position as director of veterans' affairs for the Veterans of the Vietnam War and as director of the one of Pennsylvania's Governor's Veterans Outreach and Assistance Centers, I have literally seen thousands of Vietnam veterans. I have dealt with them on a daily basis for several years, and I can assure you that their complaints and concerns are not unfounded. I have seen young men die of cancer and other diseases. I have seen birth defects in our children. I have seen those mentally tortured because of what they did or saw while in service to their country. I have met with veterans from all over this country who are having problems. There is only one thing that they have in common and that, gentlemen, is that they served their country in Vietnam.

On behalf of my brothers, I want to thank this committee for giving me the opportunity to present my views. Thank you, Mr. Chairman. I would be more than happy to answer any questions at this time.

[The statement of Mr. Swartz appears on p. 277.]

Mr. APPLEGATE. Thank you, Mr. Swartz.

Mr. Milne.

## STATEMENT OF MICHAEL MILNE

Mr. MILNE. Mr. Chairman, and members of the subcommittee, thank you for the opportunity to present the views of the Veterans of the Vietnam War with respect to House bill 1961.

My name is Michael Milne. I am the national director of the Veterans of the Vietnam War, a fraternal organization throughout the United States and we are still growing. I am a Vietnam veteran who served in the U.S. Army for 7 years. I am a victim of herbicide exposure. I must wear braces on both hands, a back brace, a neck brace, a portable tens unit on my arms, photo-gray lenses and I walk with the aid of a cane.

I have been in physical therapy since 1980. The Veterans' Administration Hospital and private physicians don't know what is wrong with me, except for the fact that I have rashes they cannot diagnose, diarrhea since 1968, chronic epicondylilus, severe chronic fibrositis, discongenic degenerative cervical neck disease, respiratory problems, urinary tract disorder, polymyostis, polytendenitis, unspecified neurological conditions, hearing and eye conditions and sinusitis. I was in perfect health when I went to Vietnam. My physical problems started in Vietnam and continue to this day.

Many studies have been initiated, many studies have been thoroughly documented, but none have received the total acceptance necessary to enable your Vietnam veterans to be justly, unquestionably and deservedly recognized as disabled. Tell us, how much longer will we have to continue to bear the burden of proof? You are here today to decide that. The fact that it has not been decided by our Government how toxic dioxin prevents Vietnam veterans from getting proper treatment and compensation. No one disputes the fact that herbicides were used. What is disputed is the level of the herbicide's toxicity. What the Veterans' Administration demands is conclusive evidence.

Dioxin is the most toxic organic compound known, producing a wide range of organ and metabolic dysfunctions. There is a general consistency between the pattern of chronic toxicity induced in animals by dioxin and dioxin-contaminated chlorophenolic compounds and those observed in exposed human populations. Such toxicity includes chronic hepatic dysfunctions, disturbances in lipid and porphyrin metabolism, neurological abnormalities, neurological disorders, glandular disorders and diseases, and birth defects, miscarriages, and still births. How much of a warning do you need? The women in Oregon were studied for 5 years. Birth defects coincided with the use of dioxin without a doubt.

Look at Vietnam veterans. Don't you think it a little suspicious that veterans whose average age is only 35 are suffering from cancers and other severe metabolic and physical dysfunctions and producing deformed children at rates far and above "predicted averages," who are experiencing illnesses more properly assigned to much older people? Don't you find that curious. And what in God's name do Vietnam veterans, people on farms in Oregon, the streets in Times Beach, Mo., factory workers in Italy, cleanup workers in Sturgeon, Miss., have in common? How about considering exposure to dioxin? How much more do we need before you start to wonder? I stand here before you as an example of a herbicide victim. I have tried to express some vital statistics and to show you the human side.

I would like to thank Mr. Daschle and the other cosponsors for taking the time and effort and showing their concern for the plight of the Vietnam veteran. I am asking that you expand H.R. 1961 to include the diseases that have been aforementioned. My brothers and I thank you for allowing me to testify.

[The statement of Mr. Milne appears on p. 281.]

Mr. APPLEGATE. Thank you, Mr. Milne.

Mr. Roxby.

## STATEMENT OF JAMES ROXBY

Mr. Roxby. Mr. Chairman and members of the subcommittee, my name is James Roxby. I am one of the original founders of the Veterans of the Vietnam War and am involved in the nationalization of the organization

I am a Vietnam veteran. I sprayed agent orange in Vietnam. I carried it on my back and sprayed it. I was hospitalized in Vietnam after being inadvertently sprayed. It is in my records right here.

H.R. 1961 will help a few Vietnam veterans the way it is now structured, but it won't help me and other Vietnam vets in my condition. Gentlemen, I am dying.

If there are any questions, I would be more than happy to answer them.

[The statement of Mr. Roxby appears on p. 284.]

Mr. Applegate. Thank you very much, Mr. Roxby.

Thank you all for appearing before the committee with very indepth and sensitive statements.

I might state at this point that I have been informed that Mr. Frank McCarthy, who was to have appeared before you, is ill and unable to appear before us today. Without objection, his statement will be entered into the record.

[The statement of Mr. McCarthy appears on p. 466.]

Mr. Applegate. Again, I would like to thank you very much.

I would like to ask you a question, Mr. Swartz—you are familiar with the Ranch Hand study are you not?

Mr. Swartz. Yes, sir.

Mr. Applegate. We understand that this study is near completion. That study has been going on for some time. We will possibly be receiving some preliminary [illegible] from that study in June. Do you have any knowledge of the study itself? What do you feel about the Ranch Hand study? Do you [illegible] it is going to provide information which may be considered [illegible]ingful? Are you familiar enough with it that you can comment on it?

Mr. Swartz. I believe I am familiar enough to comment on it.

I do not believe that it will give to us the information that is necessary. I hope that it will give us the information that shows the Vietnam veterans are suffering a wide range of medical problems. However, the people who are being studied, as the commander for the VFW stated earlier, you have to take into consideration that these people were basically the individuals who were in the rear areas who had facilities to take showers if they were sprayed with dioxin or agent orange or any other herbicides that were over there. They were able to go to a shower and immediately remove it from their bodies.

Those individuals who served in the field were not afforded that same opportunity. So, therefore, I don't feel that the people being studied in this are going to give us a true sampling of the Vietnam veterans who are having problems. I don't believe we are going to see the kind of results that we believe should be found.

I am not sure on this myself whether all individuals, too, in the ranch hand operation were supposed to be contacted. We have a gentleman who lives in our area in northeastern Pennsylvania who was not contacted. So, whether the study is going to include all individuals involved in the ranch hand operation, I don't know.

Mr. APPLEGATE. I note in your statement that you have some serious reservations about the portion of the bill that would allow the Administrator to include other diseases.

Mr. SWARTZ. Very definitedly.

Mr. APPLEGATE. And that you suggest we read the General Accounting Office report. Could you briefly summairze what that report states?

Mr. SWARTZ. Yes. This is the report here. Basically, it tears apart the VA's handling of the whole agent orange issue.

In the report—if I may just find it here—it gives a breakdown of the 14 medical facilities that were investigated by GAO when they were compiling the information for this report. Part of the categories that are listed here are given a percentage rating from 0 to 100 on 4 different areas of the study. There are VA facilities in here that have a rating of zero, as far as their overall work with the Vietnam veteran. One of these facilities, a facility in California—there is only one facility that comes anywhere near being respectable, and that is the VA hospital in Wilkes-Barre. Here we are only talking also about administrative handling of the study; we are not talking about the medical care that is being given.

Also, in here it quotes from six different physicians who believe that the only reason the agent orange study is being conducted at all is to pacify the Vietnam veterans. The physicians state it themselves which is quoted in here that they believe it was being used by Vietnam veterans for their own personal gains. These are the physicians who we have to send people to to be taken care of. I certainly don't want a physician who has that attitude taking care of my medical problems.

So the VA's handling of the whole agent orange issue has been a catastrophy from the beginning. The GAO report states that. I am glad to see the CDC being awarded the contract in order to do the epidemiological study. However, we are still talking 1991 before any conclusions can be made in that study. What do we do with the Vietnam veterans who are having serious medical problems that are not listed in H.R. 1961?

Mr. APPLEGATE. Thank you, Mr. Swartz.

The gentleman from Ohio, Mr. McEwen.

Mr. MCEWEN. Thank you, Mr. Chairman.

Did any of you gentlemen handle the dioxin, agent orange directly?

Mr. ROXBY. Yes, sir, I did.

Mr. MILNE. I also did.

Mr. MCEWEN. Could you describe to me how that worked?

Mr. ROXBY. When I first went over to Vietnam, part of my job was to help clear out an area about 8 miles outside of the port city of Qui Nhon. We had some heavy equipment on the low-lying grass. We whacked them down and we also sprayed. We sprayed it out of the back of a deuce and a half, a 2½-ton truck, with a high-pressure hose.

Once we strung up our consertina wire for the perimeter, part of my job was strapping that tank on my back and going out where the trucks couldn't go out. We referred to it as an Indian tank.

Once in Vietnam I was walking on the outside of the wire, and a truck came by and hit me with a full blast right in the face. I lost my breath, and I was bleeding in the nose and my eyes. I thought my lungs were on fire. They put me in the hospital for that one.

The second time I was hit, I was out with the tank on my back. My buddy tripped a mine. I got flipped in the air into the concertina wire. When I woke up in the hospital, I didn't have any skin on my face or any part of my upper body. Until today, I carry sores on me, on my fingers—what is left of them—on my elbows and on my shoulders. I have a rash on my groin and on my legs. Yet, there is nothing wrong with me.

Mr. **McEwen**. Was this to be diluted in any manner?

Mr. **Roxby**. Supposedly. We used it straight out of a 55-gallon drum with an orange ring right in the center of it.

Mr. **McEwen**. What was the recommended dilution ratio?

Mr. **Roxby**. There wasn't any, just like there wasn't any protective clothing handed out or any warning given to us when we did use it.

Mr. **McEwen**. There was nothing on the 55-gallon drums that said this was to be diluted?

Mr. **Roxby**. Nothing.

Mr. **McEwen**. Mr. Milne, do you have any objection to the official VA records in your case being brought before the committee for the hearing record?

Mr. **Milne**. No, sir.

Mr. **McEwen**. Thank you very much.

Thank you, Mr. Chairman.

Mr. **Applegate**. The gentleman from Texas, Mr. Hall.

Mr. **Hall**. This is probably the most cogent testimony I have heard because we are dealing for the first time, as far as I know, with people who actually handled this dioxin.

Mr. Milne, I would like to ask whether you sprayed this actually yourself as did Mr. Roxby?

Mr. **Milne**. No, sir, I did not spray it. I handled it and transported it.

Mr. **Hall**. Do you know whether or not those who did handle it and actually sprayed it did it in conjunction with a platoon of men? How was it done?

Mr. **Milne**. Generally, I can tell you how we did it. We went from Vung Tau, when we were transferred, down to Vinh Long, much further down into the delta. When we first went down to check the area out, of course, it was full foliage, completely covered. We went to check our area out where we were going to have another base camp. Then we went back down, and I did not physically spray the agent orange.

Mr. **Hall**. Did you see it being sprayed?

Mr. **Milne**. Yes, I was in the area. I was in the party that went down.

Mr. **Hall**. Was it set up by a squad of men or a platoon?

Mr. **Milne**. The tank trucks and the Indian tank style, as Mr. Roxby used.

Mr. **Hall**. Mr. Roxby indicated that he took some on his back when he sprayed.

Mr. **Milne**. Yes, sir.

Mr. **Hall**. You saw that going on?

Mr. **Milne**. Yes, sir, that is how we cleared the area, the Indian tank and the use of the tanker trucks.

Mr. **Hall**. All the people with whom you were associated with, that you saw using this, as Mr. Roxby has testified, did all of those people sustain the same type of injuries that you have sustained and Mr. Roxby has sustained?

Mr. **Milne**. I couldn't tell you that, sir. I am doing my best to try to find the people that I served with in Vietnam, but I have been having no success whatsoever.

Mr. **Hall**. Mr. Roxby, could you answer that question? Do you know of any people you served with in that area that you talked about who sustained the same injuries that you have sustained and that you are suffering from today?

Mr. **Roxby**. Again, I am having problems locating the people I was with, too. There were only 18 guys in the entire outfit. When I was down here for the last time for the Vietnam Memorial dedication, I put my name on the computer. My outfit didn't even exist.

Mr. **Hall**. I didn't understand that.

Mr. **Roxby**. My organization—I was with the 505th Air Tactical Unit—there are no records of that outfit ever existing in Vietnam.

Mr. **Hall**. You say that there is no evidence of the 505th outfit ever existing in Vietnam?

Mr. **Roxby**. Just like my medical records from Vietnam, too.

Mr. **Hall**. You have no medical records from Vietnam?

Mr. **Roxby**. I have one piece of paper stating my being injured over there and then winding up with an infection, and I think that was a mistake when they sent it to me.

Mr. **Hall**. Of course, if that situation is true, that is a disgrace on the part of somebody.

Mr. Swartz, have you made any determination as to whether or not any of the Vietnamese who were doused with this substance have come up with the same injuries that Mr. Roxby and Mr. Milne have come up with?

Mr. **Swartz**. There was a symposium—and I believe the information was just released—that was done on the Vietnamese people. I don't have access to that at this time and the conclusions of that. As far as other people while I was in Vietnam, the Vietnamese people themselves being exposed and having medical problems, I wouldn't have been aware of it, obviously, since we weren't aware of any risks.

Mr. **Hall**. Do you know whether or not any tests have been made by the U.S. Government or the Government of Vietnam as to whether any Vietnamese people who were sprayed with dioxin have sustained the same injuries that these two gentlemen, Mr. Roxby and Mr. Milne have sustained?

Mr. **Swartz**. Specifically the problems that these individuals have? No.

Mr. **Hall**. Any similar problems of any other veteran?

Mr. **Swartz**. I do believe that one of the reasons the U.S. Government stopped spraying the herbicides in Vietnam was due to complaints by the Vietnamese Government regarding various liver cancers that were being developed amongst the Vietnamese people and certain birth defects that were occurring within the population of South Vietnam.

Mr. HALL. Did some of the officials of Vietnam raise those questions at the time it was being sprayed?

Mr. SWARTZ. To my knowledge, yes, sir.

Mr. HALL. During the time that the war was in progress?

Mr. SWARTZ. In 1970, I believe, sir.

Mr. HALL. Thank you. Thank you very much for all your testimony.

Mr. APPLEGATE. The gentleman from South Dakota.

Mr. DASCHLE. Thank you, Mr. Chairman.

I want to commend all three of you for providing us with what I think to be, as Mr. Hall said, very cogent testimony. It is always, I think, very helpful to get hands-on information, the kind that only you can provide.

I would like to follow up on Congressman Hall's question. Within your organization, Mr. Swartz, are there additional Mr. Roxbies?

Mr. SWARTZ. Yes, sir.

Mr. DASCHLE. I think what Mr. Hall was asking was, is this an individual case? Is this the only case you can present before the committee that exposure to Vietnam has demonstrated?

Mr. SWARTZ. I will let Mr. Roxby answer that.

Mr. ROXBY. In the VA Hospital alone in Wilkes-Barre, Pa., there are four cases diagnosed of exactly what I am dying from.

Mr. DASCHLE. You are saying there are four cases in just one hospital diagnosed that are identical to yours?

Mr. ROXBY. That is what I know of because they are personal friends of mine.

What I would like to know is how many other ones.

Mr. DASCHLE. Do they show similar experiences in Vietnam?

Mr. ROXBY. I just buried my buddy 3 weeks ago. The other guy did have the rash on him when he left Vietnam.

Mr. DASCHLE. He had chloracne when he left?

Mr. ROXBY. Right. Right now—I was in Vietnam before him—and right now, his hands are being twisted like mine, and the rest of his body.

Mr. SWARTZ. As I am involved in this organization and as director of the Veterans Outreach Center in Wilkes-Barre, I do have occasion to see many Vietnam veterans. The condition of Mr. Roxby and Mr. Milne is not unique. Thank God many of the individuals are not in that severe a condition as Mr. Milne and Mr. Roxby. However, there are some in far more serious condition, and thousands in conditions that are not as bad but still progressing. It is not uncommon to see Vietnam veterans having medical problems.

Mr. MILNE. Mr. Daschle, it would be very easy for us to produce people for anyone to look at.

Mr. DASCHLE. So you are saying that what Mr. Roxby and Mr. Milne are presenting, Mr. Swartz, is representative of many other cases you have seen from your experience in Pennsylvania?

Mr. SWARTZ. Not only in Pennsylvania. It is definitely not limited to Pennsylvania. It it throughout the country, yes.

Mr. DASCHLE. Mr. Chairman, I understand that there are others in the audience. Maybe at some point, possibly at the end of the hearing, we can ask them to stand, because I think it would be interesting to have them identify themselves. I know that there are others who are here because of a personal interest they have in the illnesses that we are talking about today.

Again, you have provided us with very, very important testimony, and I am very grateful to all three of you. Thank you.

Mr. **Swartz**. Thank you.

Mr. **Milne**. Thank you.

Mr. **Applegate**. Thank you, Mr. Daschle.

Would the gentleman from Pennsylvania care to make a comment?

Mr. **Harrison**. Yes, I would. Thank you, Mr. Chairman. I want to thank the committee again for taking the time to hear these gentlemen.

I would like to pay a special tribute, if I can, to Mike and to Jim. It is sometimes the comfortable and inviting thing to do to hide your pain privately and not to share it publicly, and that you gentlemen are willing to come here today and make this statement, not only in your words, but in your presence, I think is a tribute to you and I am grateful to you and proud of you.

Thank you.

Mr. **Swartz**. Thank you.

Mr. **Applegate**. Does counsel from either side have any questions they would like to ask?

Mr. **Moon**. No, Mr. Chairman.

Mr. **Applegate**. Gentlemen, thank you very much. We truly appreciate your coming and giving your testimony to the committee. It certainly is going to play a very important part in assisting us in reaching our final conclusions.

Mr. **Swartz**. Thank you, Mr. Chairman.

Mr. **Milne**. Thank you, Mr. Chairman.

Mr. **Applegate**. Next we have Mr. Stephen Edmiston, associate deputy, national legislative director of the DAV.

Mr. Edmiston, we have your statement. You may read it in its entirety or you may summarize. We are under the 10-minute rule. You may proceed.

## STATEMENT OF STEPHEN L. EDMISTON, ASSOCIATE DEPUTY NATIONAL LEGISLATIVE DIRECTOR, DISABLED AMERICAN VETERANS

Mr. **Edmiston**. Thank you, Mr. Chairman. It is a pleasure to be here this morning.

On behalf of the 760,000 members of the Disabled American Veterans, I wish to thank you for the invitation to participate in today's hearing to discuss the adjudication of VA claims related to herbicide exposure by veterans of the Vietnam era.

Since my written statement has dealt with the subject of today's hearing in length, I would like to express our views on how we feel this issue can best be addressed.

As you know, Mr. Chairman, much has transpired, but little accomplished, since the issue of the adverse health effects from herbicide exposure among Vietnam veterans gained national attention some 5 years ago. To date, Vietnam veterans and their families, the Congress, and the American public are still awaiting the results of the study mandated by Congress more than 3 years ago.

Because we, too, share the concerns of Vietnam veterans and their families over the possible effects exposure to agent orange may have had upon their health, the DAV is disturbed, as most of these veterans are, by the lack of decisive action on the part of the Veterans' Administration to complete the study.

The time has arrived, Mr. Chairman, for the VA to move forward and to take positive steps to equitably consider and resolve the pending disability claims filed by Vietnam-era veterans relating to phenoxy herbicide exposure. In this connection, Mr. Chairman, we would strongly recommend, in light of the findings contained in the GAO report entitled "Health Effects of Exposure to Herbicide Orange in South Vietnam Should Be Resolved," dated April 6, 1979, that the VA be required to develop concise and indepth guidelines for the disposition of phenoxy herbicide-related claims since, as GAO reported, the toxic effects of such herbicides has been reported by humans exposed during industrial accidents involving the production of such chemicals.

In our view, such guidelines should embrace: One, the fact that veterans served in Southeast Asia during the time phenoxy herbicides were in use by the military service; two, the veteran is indeed suffering from a condition which would be compensably disabling; three, that strict adherence to the provisions of section 313, title 38, United States Code regarding the absence of any other intervening cause which may have led to the veteran's present disability; four, a measure of the level of dioxin retained in the human body which, in and of itself, would be considered the probable cause of the present disease or disability; five, incorporate all known health effects of phenoxy herbicide exposure as reported by GAO in their April 6, 1979, report, pages 17 and 18, which sets forth a variety of dermatological, internal, neurological, and psychiatric disorders; and, finally, the relationship of acute symptoms appearing immediately following exposure to more chronic, latent conditions.

Mr. Chairman, the development and implementation of such guidelines will go a long way toward insuring the integrity of the VA's disability compensation program, as well as establishing a formal procedure for the disposition of those disability claims attributed to phenoxy herbicide exposure in Southeast Asia.

This approach, in preparation of obtaining the results of the study mandated by Public Law 96-151, is by no means new. Similar guidelines were developed for claims filed by former prisoners of war—the VA adjudicated cases based upon specific guidelines until Congress received definitive study results. Subsequently, legislation was initiated and enacted into law which generally liberalized the various VA benefits and services provided to former prisoners of war.

Mr. Chairman, I trust the subcommittee will give serious and favorable consideration to our views on this important subject.

That concludes my testimony, Mr. Chairman. I again wish to thank you and the members of the subcommittee for permitting us to provide our views on this important matter.

[The statement of Mr. Edmiston appears on p. 285.]

Mr. APPLEGATE. Thank you, Mr. Edmiston. I respect and appreciate the very courageous statement that you have made.

I realize that when this resolution was adopted, it was probably not met with overwhelming acceptance and was possibly very unpopular with some of your members.

How many Vietnam veterans do you have in your organization?

Mr. EDMISTON. Of our total membership, approximately 30 percent are veterans of the Vietnam era.

Mr. APPLEGATE. Yes.

Mr. EDMISTON. I would also add that the people who are employed as DAV national service officers throughout the United States who assist veterans filing claims with the Veterans' Administration as well as other Federal agencies; over 90 percent of these employees are disabled veterans of the Vietnam war.

Mr. APPLEGATE. To what extent do the members of your organization participate in the formulation of policy, and how many members participated in the formulation of this particular policy?

Mr. EDMISTON. The resolution was adopted unanimously at our last national convention. I, in all honesty, couldn't tell you the number of Vietnam veterans who were present at that convention, though I know the number was significant and it has been a number which has continued to grow through the years.

Mr. APPLEGATE. Could you define what you mean by unanimously? Was it just a voice vote?

Mr. EDMISTON. It was a voice vote. There was no objection to the resolution.

Mr. APPLEGATE. Was there objection to it while the policy was being formulated and, if so, by whom?

Mr. EDMISTON. If there was objection, I did not hear it or know of it.

Mr. APPLEGATE. I mean during the period of formulation of the policy.

Mr. EDMISTON. The resolution came to the national convention from one of our State departments which, in turn, it had come up through the network that we have from one of our chapters. If the committee would like, I think I could probably investigate and determine exactly where the resolution originated. But, to my knowledge, that resolution came from the grass roots, from the membership of the DAV.

Mr. APPLEGATE. Thank you very much, Mr. Edmiston.

The gentleman from Ohio, Mr. McEwen.

Mr. MCEWEN. Thank you, Mr. Chairman.

Mr. Edmiston, you stated that, of your 300 DAV service officers, all but 25 or 30 of them are Vietnam veterans, and most of whom are seriously disabled. Would you speculate on how they feel about this matter of presumptive service connection for any disability? Have you discussed it in that manner with them?

Mr. EDMISTON. Myself, being a former national service officer and representing veterans and the staff here in Washington, D.C.—the legislative staff are all former national service officers—we have discussed, not only among ourselves, but with some of our national service officers in the field, and they agree, that to establish a presumption as H.R. 1961 proposes, it would deviate from the history of the compensation program throughout the years. That is, in terms of the lack of a factual basis upon which to make that presumption.

Mr. McEwen. Have you seen or has your organization noted soft tissue sarcoma cases presented to the Board of Veterans Appeals which, in the DAV's professional service officers' judgment, have been disallowed when the prevailing evidence would have caused a different decision?

Mr. Edmiston. I am sorry, I don't quite understand your question.

Mr. McEwen. Do you feel that assistance was denied by the board, even though the evidence as to the injury was in a preponderance? Do you feel that there is a prejudice against either soft tissue sarcoma or the other similar or related injuries relating to agent orange exposure?

Mr. Edmiston. These would be claims based upon exposure to the herbicides? I am assuming that is what you are referring to.

Mr. McEwen. They wouldn't be stated as such, but that would be the natural understanding.

Mr. Edmiston. We have represented veterans who have filed claims for soft tissue sarcomas based upon other related matters, but not specifically to the herbicide. We have represented some of those cases. I am not familiar personally with any of them.

Mr. McEwen. Thank you again for your testimony, Mr. Edmiston.

Mr. Applegate. The gentleman from South Dakota.

Mr. Daschle. Thank you, Mr. Chairman.

Mr. Edmiston, let me ask you a couple of questions. Do I understand the DAV's position to be in opposition to H.R. 1961?

Mr. Edmiston. We do not favor its passage.

Mr. Daschle. I guess I am puzzled, because your resolution says that your organization seeks the enactment of legislation which would extend open-ended presumption of service connection for any identifiable residual disability or disease incurred by veterans which is scientifically proven to be the result of exposure to agent orange. How do you resolve this difference?

Mr. Edmiston. We believe that due to the division in the scientific community over the issue—as to whether or not the long-term health effects from exposure to the herbicide—is how we resolve the difference.

Mr. Daschle. Do you agree with Mr. Walters' statement that the medical community has reached a consensus that the causal relationship exists between the exposure to dioxins and chloracne and exposure to dioxins and porphyria cutanea tarda?

Mr. Edmiston. The information and literature that we have had available to us does indicate that the investigations that have occurred as a result of industrial accidents where individuals were exposed during those times, there have been reported instances where these various problems have resulted in individuals who were exposed.

Mr. Daschle. Then you agree with Mr. Walters' statement that the consensus does exist on these two things; is that not correct?

Mr. Edmiston. There is a consensus in that instance, yes.

Mr. Daschle. I am puzzled, I guess, by what you just said. You just said that where there isn't a consensus, we can't agree that there is a presumption. Now you have said that there is a consensus on these two and, yet, you don't favor this legislation. How do you resolve that?

Mr. EDMISTON. I think what I am referring to, Mr. Daschle, is the fact that in those instances where individuals were exposed to the chemical in industrial situations differs from that to the long-term effects of exposure. That has not been cleared. I think it has been proven, the fact that the skin disorder and the liver disorder have been conditions that have appeared immediately following exposure to the chemical.

Mr. DASCHLE. I beg to differ with you. That isn't the case at all. We have seen long-term effects that have only been evidenced well after exposure. I guess what you are saying here is you are drawing a distinction between cases of exposure in this county and cases of exposure in Vietnam. I am really troubled that a member of a major veterans organization would draw sort of a double standard in that way.

Are you familiar with MS which, of course, is a presumption, or the cardiovascular presumption that the VA so far has been willing to grant? What do you think about the fact that there does seem to be a difference of opinion on the MS or the cardiovascular presumptions, yet a presumption in both cases has been established. How do you feel about the distinction between the two diseases?

Mr. EDMISTON. I think that each disease, MS or—as you were referring to earlier, the relationship between amputation and cardiovascular disorders—I don't think one can draw a specific distinction between them.

Mr. DASCHLE. You can, though, in that the study did not determine the cause of the increased risk of cardiovascular disease. That is the same problem we have here. We have no cause determination. Yet, we see the relationship, the higher incidence. On that basis—and I emphasize again to the committee, this is really an important point—we made this decision on compensation on the basis of one internal study of a medical followup agency of the National Academy of Sciences. That was the one study upon which we made a presumption of greater disability.

Now what the DAV is telling us is that we need a lot more studies for agent orange. I beg to have you explain to me the difference, if you will.

Mr. EDMISTON. First of all, we are not asking for more and more studies. I think the studies that are underway at the present time should provide sufficient information to reach a reasonable conclusion on the issue.

Mr. DASCHLE. I guess we could go on and on. I am puzzled by the difference that really seems to be striking between the resolution which calls for this legislation and your opposition of this legislation which, obviously, is based on the statements already made by Mr. Walters' that, of course, there is a consensus of scientific information on two of the three presumptions in H.R. 1961.

We could have extended this thing a mile wide to contain all kinds of diseases mentioned by the witness prior to you, but we chose not to. We chose to tighten it, to make it as conservative as possible, so that we would not have the kind of opposition presented by your testimony. So I am, frankly, very disappointed.

But I certainly respect your opinion. I hope that you are right and that it does represent the majority view within the DAV. I will be speaking to my State convention this weekend, and I am certainly going to be bringing this up then. Thank you very much.

Mr. **Edmiston**. Mr. Daschle, if I may add that if you would closely examine our suggested guidelines for the VA to consider these claims, that in taking into account the different things you brought out today, I think the bottom line would be that the VA could resolve many of those claims in favor of these veterans. I don't think there would be any problem.

Mr. **Daschle**. Mr. Chairman, would you mind if I had one more minute?

Mr. **Applegate**. Go right ahead.

Mr. **Daschle**. Again, I am puzzled then about what you just said. You say that you support giving the veteran the benefit of the doubt, that they should be able to resolve these things given the benefit of the doubt. But, yet, you oppose putting that in writing. When we limit the benefit of the doubt simply to three diseases, two of which the Administrator clearly shows to represent the consensus in the scientific community—how do you—you make conflicting statements that are very troubling to me. I know you are well intended, and I am trying to figure out just where you are coming from.

Mr. **Edmiston**. I don't think we are trying to be conflicting in our statements. We feel that the guidelines that we have set forth here broadens the scope, first of all, in terms of permitting the VA to take into account many different disabilities and impairments that veterans experiencing.

Mr. **Daschle**. Let me ask you one final question. I know that there are many witnesses waiting to testify.

In your point of view as a national service officer, as one who has handled claim after claim after claim, would it not, in your opinion, be to the advantage of the veteran to have clearly delineated in law the presumption of doubt with regard to these three diseases when a veteran comes before the VA to seek compensation?

Mr. **Edmiston**. That would be ideal.

Mr. **Daschle**. Thank you very much.

Mr. **Edmiston**. But I wouldn't necessarily agree.

Mr. **Applegate**. Mr. Edmiston, I have just one quick question.

I am trying to figure out after which scientifically proven or disproven study are we talking about that you would make a final decision. Is it the Swedish, the Australian, the Ranch Hand, or the one that we mandate the Veterans' Administration to do.

Mr. **Edmiston**. The one mandated by Public Law 96-151.

Mr. **Applegate**. Thank you.

Mr. **Edmiston**. Thank you.

Mr. **Applegate**. Does counsel have any questions?

Mr. **Moon**. No questions.

Mr. **Applegate**. Thank you very much, Mr. Edmiston. I appreciate your appearance before the committee.

Next we have Mr. Jack Powell, who is the executive director of the Paralyzed Veterans of America.

Jack, you may proceed as you wish.

## STATEMENT OF R. JACK POWELL, EXECUTIVE DIRECTOR, PARALYZED VETERANS OF AMERICA

Mr. POWELL. Thank you, sir.

I knew when the PVA's legislative director asked me to give this testimony that something must be wrong. I find out that I walked into a lion's den perhaps.

It is always a pleasure to come meet with the members of the committee and with the staff. It is not necessarily always a pleasure to have to disagree with a bill proposed by someone we think as highly of as Mr. Daschle. Nevertheless, we have submitted a statement for the record. Recognizing the time restraints of the committee, I will try to be very brief.

PVA, for 37 years, has taken an intensive interest in medical care provided by the Veterans' Administration. We have certainly pushed for many of the benefits that are presently provided, including a number of the presumptive benefits, such as MS presumptive period, and have testified before this committee and others on many occasions about the possibility of extending some of those presumptive periods.

Nevertheless, PVA does have a great degree of difficulty with H.R. 1961. The term "may be the result of exposure" is, to say the least, extremely broad and extremely vague. It has been our feeling acquired over the years that veterans do substantially better by putting their fate, as it were, in the hands of the committee and in the hands of Congress. So another portion or problem with the bill is leaving the determination up to the Administrator of the VA.

Certainly we don't have any problems with Mr. Walters, who we think very highly of in his initial outings as Administrator of the VA. However, we recognize that there are many pressures—not merely from the committees and from the Congress, but from Presidents and folks within the administration and others—that come to bear on the Administrator. So we have some great budget considerations and that type of thing. We have some great concerns about allowing this tremendous flexibility to the Administrator.

Insofar as the three diseases specifically specified in the bill, according to the best figures provided by people within the VA and people within the medical community, we are talking, at most, about 3,000 people of the 106,000 Vietnam veterans who have taken the agent orange examination at the VA.

If in fact there is a health related problem which still remains to be scientifically proven, then perhaps 3,000 is mere tokenism. Perhaps it is far broader. But we don't think it is appropriate for this committee or for this Congress to legislate in answer to a scientific question to which the evidence is not in. We believe, as do our brothers in DAV, that upon the conclusion of a congressionally mandated study, that a determination should be made and, based upon those facts, whatever is appropriate should be provided to veterans. Certainly PVA will be before you at that time to argue for the appropriate benefits and the appropriate medical treatment.

We do believe that the present position of the Veterans' Administration, at least as articulated by the VA Central Office, to provide treatment for agent orange, medically, is an appropriate position. But we do not believe that it is appropriate to provide compensation on the evidence that exists today.

That, basically, is it. It would be a lot easier to testify "yes." It probably would be a lot more popular. It might even increase our membership a little bit. Fully a third of PVA's members are Vietnam veterans. I am a Vietnam veteran myself. But I don't think that we can let emotionalism or frustration or anxiety set the tone of veterans compensation laws, because it could equally and more probably go against us as it can work for us.

Thank you, sir.

[The statement of Mr. Powell appears on p. 296.]

Mr. APPLEGATE. Thank you very much, Jack. It is always a pleasure to have you before the committee because you are always straightforward and very articulate. We always appreciate your testimony.

I note that your basic position is pretty much in agreement with the DAV in that you personally feel that sound medical principles should be the yardstick on which the benefits should be measured.

What we are considering now is just how sound the arguments are that there is a relationship between exposure and certain disabilities. I have just a couple of questions.

Does the evidence show that the Vietnam veteran is suffering from the conditions mentioned in this bill to a greater degree than other veterans who did not serve in Vietnam? What perhaps should we do about the provision in the bill that would include veterans who served in Thailand or other countries in Southeast Asia where herbicides were not used? And should we try to put some limit on the time of service in Vietnam in relationship to the exposure? On that same line, what about the temporary service of veterans who were there maybe for a week or 10 days?

These are the questions we have to ask ourselves before we arrive at any conclusions, and the sources of information which we utilize will certainly be varied. I think they are very important.

Mr. POWELL. Mr. Applegate, I was in St. Louis when Anne Burford came down to tell the folks that she was going to buy them out. I guess, as a Vietnam veteran, my immediate response was one of intense frustration and anger, and I said, "My God, if I have ever seen a classic example of a double standard, here it is, here it exists. If they treated the people in Times Beach the way they treated Vietnam veterans," I said to myself, "the people in Times Beach would grow to old age still living in the same location waiting for a decision to be made."

And then I recognized one very real difference. Veterans who served in Vietnam and were exposed to dioxin—and I was myself—are no longer suffering that exposure. They may be suffering from problems—that is what we need to determine—they may be suffering from problems that resulted from that exposure, but they are not day in and day out being reexposed. I think that is why there is a very real basis for a differentiation between the way we treat the folks in Times Beach, and I understand there are 17 other areas in the State of Missouri alone which have high levels of dioxin exposure. That is a very real reason to treat those folks differently. You have to act now. You don't have time to answer the questions. But the exposure of Vietnam veterans is over, for the good or for the bad of it. Now we need to ask some questions.

I don't know of any law that has been passed by this committee or by Congress that is written in stone. It changes. We say that probably and logically there should be some kind of a presumptive period similar to what there is with MS and a number of other things. How long should that be? We don't know. I don't think that anybody is ready today to say that the presumptive period for MS will never ever be changed. If we find more medical evidence, I am sure that PVA will come before this committee and ask for an extension of the presumptive period for MS. I am sure that if we have the adequate evidence, this committee will listen to us and will hear us and will grant that.

I am sure that if a reasonable time period is set for the exposure effects to surface from agent orange and evidence is developed that shows that it could be 20 years instead of, say, 7, that it will be extended to 20 years.

But the questions that you point out are the questions that I don't think the information exists today to answer. I think that we do something foolish and irresponsible by playing up the fears that so many of my friends have and I have, who served in Vietnam by the attribution of potential problems that we can't scientifically prove exists because, for the 3,000 folks, that may be fine, but what about the remaining 103,000 who have taken the examination and what about the millions who served and haven't gone in? What additional questions are we going to be causing them to ask themselves?

Excuse me for getting carried away.

Mr. APPLEGATE. The gentleman from Ohio, Mr. McEwen.

Mr. MCEWEN. Thank you, Mr. Chairman.

Mr. Powell, I appreciate your excellent testimony and your obvious compassion and concern for the difficulties surrounding this situation and understanding how reasonable people can differ.

One additional question I would ask, because I don't know the answer to it, is about the Times Beach situation. Are those people receiving continuing compensation or are they moved out of their homes and are on their own?

Mr. POWELL. There is no continuing compensation for any of those that I am aware of. There was nothing proposed by EPA in that area.

What was merely done is that the property they had was purchased. I would imagine that if there are health related problems that come up, that would be dealt with probably by the Department of Health and Human Services, if there can be some evidence shown to prove it.

Mr. MCEWEN. Thank you very much.

Mr. APPLEGATE. The gentleman from South Dakota, Mr. Daschle.

Mr. DASCHLE. Thank you, Mr. Chairman.

Mr. Powell, you cited the distinction between Times Beach and Vietnam. Congressman Hall earlier posed the question to another witness that if we were a jury and we had to weigh the evidence and come up with a decision based on available scientific information, just what would you have us do?

We had such a jury a while back in a case in Illinois with railroad workers. A jury was organized and heard the evidence and, on the basis of evidence presented, made a decision that awarded 47 railroad workers with $58 million. How do you draw the distinction between those railroad workers and Vietnam?

Mr. POWELL. I don't know enough about the railroad workers' case to comment on it to any degree of authority. But I certainly would say this—and I am sure that you would have to agree with me, sir—that we have all read cases of juries' verdicts in newspapers and not necessarily agreed with the verdict handed down by the jury.

Mr. DASCHLE. How would the PVA have testified in a case like that?

Let me repose the question. Let's assume that you are called upon as a witness in one of the many trials that are now ongoing where Vietnam veterans are suing Dow Chemical, for example, for compensation as a result of exposure. How would the PVA testify in a case like that? Would you testify in favor or against the veterans?

Mr. POWELL. I think that we would ask an extension until the scientific evidence is in.

Mr. DASCHLE. How much more scientific evidence—posing the question that Mr. Applegate asked—does the PVA believe that they need, given the fact, as I have said before several other witnesses that, apparently, in the minds of the VA, the scientific evidence already has drawn the conclusion that there is a relationship between chloracne and PCT and exposure to Vietnam? Are you satisfied to draw those conclusions?

Mr. POWELL. I have heard you mention the statements by the Administrator several times, and I heard the Administrator here this morning, but if I could ask the question of you, then I would know how to answer that question. When the Administrator said that the scientific or medical community has made a determination that there is a possible cause of PCT from exposure to dioxin, did he expand at all on whether they were talking about instantaneously, or is the medical community saying that if you would drink a glass full of dioxin you are going to get PCT, or are they saying that if you want, walk through an area where dioxin was 20 years later and you will get PCT that is caused from dioxin? Did he clarify that? Can you clarify that for me, sir?

Mr. DASCHLE. He said exposure, which kind of leaves it wide open, which is all the more the point. He didn't say it had to be 20 parts per million or 200 parts per million. He said that exposure to Vietnam——

Mr. POWELL. Did he say how recent?

Mr. DASCHLE. Of course, he did; yes.

Mr. POWELL. How recent the exposure was?

Mr. DASCHLE. He said that there is a difference. Of course, that is the point.

You are avoiding the answer. I think the point is clear. When you can show a causal relationship, when you can show a definite link, there ought to be compensation. I think the PVA would say the same thing the DAV said—I would be surprised if you would say no to this, but you might. Would it not, in your opinion, be more advantageous for a PVA member to come before the Veterans' Administration with this presumption in writing if he was to ask for a presumptive disability based on the exposure that was discussed by Mr. Walters?

Mr. Powell. As a former service officer, Mr. Daschle, I would say that anytime that I represented a veteran before the Veterans' Administration, I always look to see if I could find something in writing obligating the VA. That was a significant portion of the battle.

Mr. Daschle. That certainly answered the question. That is the whole point behind the legislation. It really makes me wonder why you oppose the legislation if that is the case. We know that, to the satisfaction of the VA, two out of the three diseases already show the causal relationship. We know that by putting those in writing it would be easier for an organization such as yours to come to the Veterans' Administration for compensation. We know that, on the basis of other diseases—for example, the cardiovascular disease—I doubt very much that PVA would advocate the repeal of that additional compensation based on the fact that we have only had one study, based on the fact that there is no determination that the cause of the increased risk of this disease can be attributed to amputation.

We have all this, and yet we get a double standard that I find confusing, to say the least. But as Mr. McEwen said, we are all certainly well intended, and we can differ on these issues. I appreciate the difference, and I respect your opinion. I am glad you are here.

Mr. Powell. I thank you, sir. I am sorry that you get the impression of a double standard. I may be extremely naive—I know Mack Fleming has accused me of that from time to time—but I don't see the double standard. I just do not see it.

We think that this committee and the Congress, in its finer wisdom and judgment, after viewing many, many questions about agent orange, decided that the best thing to do was to have a scientific study conducted. We do have some problem with the fact that it took so long to get the damn thing started, and we do have some problems with the fact that, halfway through part of the process, now we turn around and pass it on to CDC. I guarantee you that we would be in here pounding the table if now CDC wants to pass it on to someplace else.

But we abided by that decision made by this Congress to conduct that study. What we are merely suggesting is that, rather than deal piecemeal with this question, we will wait until that study is completed. If Congress can legislate an answer to a medical problem, I hope that Congress will decide to legislate a cure for paralysis.

Mr. Daschle. You are an articulate witness, but you dance around the issue. The fact is that there is no question there is a double standard. The fact is that you have chosen to side with the VA against the veterans in giving them the presumption of doubt when it comes to two of these three diseases. If, as our legislation would do, we find that there is a clear-cut consensus, there is a unanimous opinion that these diseases are not caused by exposure, we have a sunset provision in the legislation. Why in heaven's name would the Paralyzed Veterans oppose legislation that simply gives the benefit of the doubt to the veterans when we have substantial evidence already existing.

You said, too, that you favor the provision based on one study which doesn't show any causal relationship between cardiovascular disease and amputation. So how can you tell me that there is no double standard here?

I see we are out of time, but I would really like to pursue this, even personally if we could, because I am still confused.

As I say, I do appreciate the information you have given us, and thank you for being here.

Mr. APPLEGATE. Thank you, Mr. Daschle.

Thank you, Mr. Powell.

Does counsel have a question?

Mr. MOON. I would like to clarify for the record what Mr. Walters said this morning about PCT. To quote directly from his statement:

> The medical community has also reached a consensus that, in some individuals, porphyria cutanea tarda (PCT), a liver disorder, may be triggered by dioxin exposure. However, no medical studies have been concluded which establish a causal connection between exposure and the initial occurrence of these disabilities long after exposure.

That is the end of the quote.

Mr. POWELL. That sounds somewhat familiar. Thank you, sir.

Mr. APPLEGATE. Thank you again, Mr. Powell.

Our next two witnesses are Mr. Phil Riggin, director, National Legislative Commission of the American Legion; and Mr. John Sommer, deputy director, National Veterans' Affairs and Rehabilitation Commission.

Gentlemen, it is a pleasure to have you back before the committee.

## STATEMENTS OF PHILLIP RIGGIN, DIRECTOR, NATIONAL LEGISLATIVE COMMISSION, THE AMERICAN LEGION; AND JOHN F. SOMMER, JR., DEPUTY DIRECTOR, NATIONAL VETERANS' AFFAIRS AND REHABILITATION COMMISSION, THE AMERICAN LEGION

Mr. RIGGIN. Thank you, Mr. Chairman.

Like several other people here this morning, we sincerely wish to commend you and the subcommittee for providing this forum on this very, very important and delicate and sensitive issue.

We are prepared to summarize our statement this morning. You have already made the necessary arrangements to have the full statement entered into the record. John Sommer will offer a summary of that statement, and we will both be available for any questions the subcommittee may have.

### STATEMENT OF JOHN SOMMER

Mr. SOMMER. Thank you, Phil.

Mr. Chairman and members of the subcommittee, the American Legion appreciates this opportunity to present our views on H.R. 1961 which, with respect to Vietnam veterans exposed to agent orange, would establish a presumption of service connection for soft tissue sarcomas, porphyria cutanea tarda, and chloracne, as well as any other disease that medical research has shown may be due to exposure to herbicides as determined by the Administrator and prescribed by regulation.

The measure also provides the Administrator with the needed authority to determine that the other diseases may be included based upon scientific evidence and to promulgate regulations relating to the additional diseases.

May we preface our summary by stating that the American Legion does not support any legislative or administrative issue because it may seem to be the popular thing to do or due to the fact that the issue may be politically attractive. The positions of the Legion are based upon our understanding of an issue acquired through existing knowledge and objective research of pertinent subject matter.

Our review has shown that there is scientific evidence that the three specific conditions contained in H.R. 1961 may well result from dioxin exposure, and examples of that evidence are set forth in the text of our full statement.

As for soft tissue sarcomas, we have included information relating to several studies, including the Swedish epidemiological case control studies which the VA find unacceptable. The American Legion feels that the evidence is adequate enough to have raised a level of suspected cause so as to be sufficient that reasonable doubt should be weighed in favor of the Vietnam veteran and service connection should be presumed for this condition.

With respect to porphyria cutanea tarda, we found a substantial number of references to the connection between dioxin and this condition, including one contained in a VA chloracne task force report. The Legion feels that available evidence is sufficient to illustrate that a causal relationship does exist and a presumption of service connection should be established for porphyria cutanea tarda.

The same is true for chloracne, which, in many instances, is also mentioned in the same literature as the PCT.

With respect to the diseases that would be listed in regulations prescribed by the Administrator as required by H.R. 1961, we have urged the Centers for Disease Control to release all relevant findings as they become available during the epidemiological study of the long-term health effects of agent orange exposure, which was mandated by Public Law 96-151.

In conclusion, Mr. Chairman, the American Legion fully supports the enactment of H.R. 1961. We understand and appreciate that the intent of this legislation must undergo the prudent deliberation of this subcommittee and, as with other issues where differences of opinion in the scientific community exist, we have no doubt that the bill will be closely but objectively scrutinized.

As Mr. Riggin previously mentioned, the American Legion thanks you, Mr. Chairman, for your timeliness in scheduling this hearing on an issue that is of great importance to Vietnam veterans.

That concludes our summary.

[The statement of Mr. Riggin and Mr. Sommer appears on p. 300.]

Mr **Daschle** [presiding]. Mr. Sommer, thank you very much for your statement. I deeply appreciate the confidence shown in this legislation by the American Legion. The leadership that they have established over the years on agent orange and on its work with Vietnam veterans is admirable, to say the least, and we are grateful to you for giving us that kind of support.

In your history in the work that the American Legion has done with presumptive disabilities and seeking compensation, has that whole question of providing compensation on the basis of presumption worked well in your situation, in your experience?

Mr. **Sommer**. We have found, as has previously been mentioned in today's hearing by other witnesses, that presumptions have been established based upon what we consider to be a lesser amount of evidence than what is available to support the enactment of this bill.

One study was mentioned with respect to the amputations and the cardiovascular disorders. We feel that the results of that study are valid. Another, which I believe you mentioned earlier, dealt with multiple sclerosis. There was much less evidence on which to base a 7-year presumption for multiple sclerosis than there is on which to enact H.R. 1961, as we see it.

Mr. **Riggin**. Mr. Chairman, may I expand that slightly?

Mr. **Daschle**. Yes.

Mr. **Riggin**. I think it is rather common knowledge that the entire concept of presumption was found necessary by Congress and by VA because of consistent lack of evidence in medical records generally over a period of two or three different wars. Evidence of medical treatment obviously is not always available in one's record when you consider wartime circumstances. It is precisely that basic lack of evidence that draws us toward the presumptive mechanism that is called for in this particular legislation.

So, I don't think that the concept of presumption or the basic reason for presumption over the years can be lost in the discussion of this entire issue of agent orange residuals.

Mr. **Daschle**. I thank both of you for your statements in this regard. I think that what we have been able to show is that we do have a substantial amount of causal relationship and scientific studies already existing which show that the causal relationship has affected Vietnam veterans. I am very pleased to see that the American Legion has demonstrated its leadership once again on this issue.

Does counsel have any questions that they wish to ask of these witnesses?

Mr. **Moon**. Mr. Sommer, I just wanted to make one comment. You rely heavily on the Swedish studies in your testimony. The VA doesn't seem to accept those Swedish studies as totally valid and there are others in the scientific community that find some fault with the way in which those studies were conducted.

The chairman has indicated that, during the course of these hearings, we want to try to get a little more information on the criticisms of the Swedish study so we will know how much reliance to put on them. I just wanted to offer that comment.

Mr. **Sommer**. Thank you, Mr. Moon. We would also be interested in hearing additional information on this, because from the material that we quoted in the statement, it was quite clear that the Environmental Protection Agency felt at that time that these studies were quite valid.

Mr. **Daschle**. I appreciate that. They certainly did. Of course, the Swedish studies are only one resource. We have a great deal of information compiled within our own country, as we will see presented tomorrow. You make reference to that as well. I think to cite only the Swedish studies would be a disservice to the tremendous amount of work done in our own country, for which we can be very grateful.

Mr. Wilson, do you have any questions?

Mr. **Wilson**. Mr. Sommer, for a long number of years, the American Legion has had some excellent doctors as medical consultants. Was their expertise and advise sought as the Legion got to its position?

Mr. **Sommer**. Our medical consultants' advice is often sought. They were certainly consulted with in this issue. However, of course, our position is based upon the resolutions that are approved by our two policymaking bodies, the national convention and the National Executive Committee, and our support for this legislation is based upon Resolution No. 410, which was approved by the 1982 National Convention in Chicago.

Mr. **Wilson**. Mr. Sommer, the other witnesses, at least some of them, have been asked for their comments about the Ranch Hand study. Would you comment on that?

Mr. **Sommer**. Yes, sir, Mr. Wilson.

We feel that the Ranch Hand study will be a very important part of the total research effort that is being done on the issue of agent orange. However, we would not want the Ranch Hand study to be the only study that anything was based upon.

The American Legion also has reservations and concerns, as have been expressed by previous witnesses, in that the individuals who were involved with Operation Ranch Hand had much better sanitation facilities available to them than did infantrymen in the field who many times were not able to take a shower or change clothes for 18, 20, or more days. We feel that this would make a big difference.

Mr. **Wilson**. Thank you, Mr. Chairman.

Mr. **Daschle**. Thank you, Mr. Wilson.

I have one remaining question that I ran out of time in asking prior witnesses from other veterans organizations. It occurred to me that I had intended to ask all of the organizations this question. Has it ever been presented to you, within the VA or within the Veterans' Committee or within any kind of advice you have been given, that by supporting compensation on a presumptive disability for agent orange, we would pay Peter by robbing Paul, that is take away compensation benefits provided to other veterans for other recognized disabilities currently within the law? Has that been presented to you, Mr. Sommer?

Mr. **Sommer**. Not to my knowledge, Mr. Daschle, and we would hope that a decision would not be based upon that.

Mr. **Daschle**. I certainly would, too. It, I think, is a very serious question, and I wish we could have asked the other witnesses. I fear that that may be part of the motivation, that they fear that, by opening this up, so to speak, that we are then going to be taking away from other veterans who are duly compensatable already.

I don't want any competition. The Budget Committeee added on an additional amount for compensation under agent orange, and they didn't say that out of that compensation budget we have to provide compensation to agent orange victims. To threaten any of the other organizations in that manner would certainly be a disservice to this bill and to agent orange victims. I am glad to hear that, at least from the American Legion's perspective, that was not an issue.

**Mr. Sommer.** To carry on that conversation a little further, I believe that you mentioned that the total cost of this could possibly be $4 million annually. We are quite aware that both soft tissue sarcomas and porphyria cutanea tarda are fairly rare conditions, so we are probably talking about a relatively small number of veterans who would be allowed claims for these conditions if this bill were enacted to begin with.

**Mr. Daschle.** The VFW was asked if expansion of the program could be supported by the VFW. If we could show conclusively that additional diseases can be related to exposure to Vietnam, is it fair to assume that the American Legion would then support inclusion of those diseases as part of the presumptive disability?

**Mr. Sommer.** Most certainly.

**Mr. Daschle.** You have been very helpful, both of you. I appreciate the kind of information you presented to us today.

Thank you.

**Mr. Sommer.** Thank you.

**Mr. Riggin.** Thank you.

**Mr. Applegate** [presiding]. Thank you, gentlemen. We appreciate your testimony very much.

Next we have Mr. Lewis Milford of the National Veterans Law Center.

## STATEMENT OF LEWIS MILFORD, NATIONAL VETERANS LAW CENTER

**Mr. Milford.** Good morning, Mr. Chairman. Thank you for the opportunity to testify on H.R. 1961.

The National Veterans Law Center has represented Vietnam veterans on the agent orange issue in Federal court and before the Federal Government for several years. We fully support H.R. 1961. We, along with the veterans we represent, congratulate Congressman Daschle for his long and frequently lonely support for this fair and compassionate piece of legislation.

This is a bill that deserves to be supported, because it is based on sound scientific evidence. But, more important, it is necessary as matter of policy because the VA has shown it will ignore scientific studies that do not support its official position that agent orange is harmless.

My statement really boils down to two words: "proof and policy." Two things are clear about the scientific evidence on the cause-and-effect relationship between agent orange and soft tissue sarcomas.

First, the public health agencies of this Federal Government, the Environmental Protection Agency, the Centers for Disease Control, and the National Institute for Occupational Safety and Health, all have stated that the European soft tissue sarcoma studies are good, valid, epidemiological studies.

These conclusions were made in the last days of the last administration, but the agencies under the Reagan administration have not withdrawn these conclusions, and we assume that they are still valid.

Probably most importantly, even the top scientists, Federal Government scientists, working on agent orange for the Health and Human Services Department said that the studies do show a correlation between phenoxy herbicide exposure and soft tissue sarcoma.

On the other two diseases, as we saw in the VA's testimony this morning, the cause-and-effect connection is conceded.

I want to make a point about the burden of proof and the standard in a court of law that was mentioned this morning. I teach a seminar in American University Law School on toxic substances litigation, and I ask my students frequently about the issue of causation in a toxic substances case.

The law is very clear that the statistical association from epidemiological studies is the functional equivalent of causation in a toxic substance case. There is no other evidence other than animal studies and epidemiological studies that can be used to demonstrate the cause-and-effect relationship.

In case after case, in Federal court after Federal court, State court after State court, it is very clear that one or two, certainly three, four, or five epidemiological studies that show a statistical relationship between exposure to a chemical and an increased incidence of disease is more than sufficient to show causation between that chemical and that disease. That is the law, and any lawyer who is handling toxic substances cases will tell you that is the fact.

Now, how is this proof handled by the VA, which is the second question. The VA has basically taken three different approaches on these scientific studies.

First, unfortunately, the VA leads the chorus of statements made by the chemical industry in the last couple of years to increase the burden of proof in toxic substances litigation, effectively, to reverse years and years of public health policy, to make fair and compassionate legislative decisions on the basis of human studies.

The VA would have this Congress wait until 6, 7, 8, 9, 10 years—presumably, until the correct studies are in. I think that is really what we are talking about.

The second position is that there is simply not enough evidence. The VA is trying to increase to an unreasonable level the burden of proof that veterans would have to establish in order to make compensation in an agent orange case.

Third, I think what we have heard and what a VA official, in fact, has said on national television, that we are really seeing another case of blaming the victim; that the agent orange issue—we heard this 5 years ago and now we are hearing it again—is a result of emotion and hysteria. They say this is not a very serious problem that Vietnam veterans were exposed to, even though it is probably the most toxic substance that has ever been created.

I think another point that has been made by Congressman Daschle, and it is in my testimony, concerns the VA presumption of arteriosclerosis disease that is connected to an amputation.

Congress ordered that the VA conduct an epidemiological study. The VA contracted with the National Academy of Sciences to study whether veterans with certain amputations have a higher incidence of heart disease.

The National Academy of Sciences issued one study, and that study said very clearly that the agency could not determine the cause of the increased risk of disease. In fact, I will quote from the study.

The report concluded, and I quote, "The study was not designed to test specific hypotheses about the causes of the increased risk."

As a result of that study showing only increased risk, no cause, the Veterans' Administration issued a regulation that created a presumption of heart disease.

So I think what we have here is not only a VA disavowal of previous policy, but in fact, an attempt to increase the burden of proof over and above what a court of law would require and over and above what the VA itself has required in a similar case of service connection.

I want to make one last point. I think it is unfortunate that the VA Administrator was not here to answer questions. I hope that we don't have a case of the new head of the agency running from the truth, like the agency has done for the last 4 or 5 years.

That concludes my testimony.

[The statement of Mr. Milford appears on p. 311.]

Mr. **Applegate**. Thank you very much, Mr. Milford. We appreciate your testimony.

The gentleman from South Dakota.

Mr. **Daschle**. Thank you, Mr. Chairman.

I really want to commend you for an excellent statement. I just wish this committee was filled this morning to listen to a very reasoned presentation.

Sometimes I get so emotionally attached that it is hard to put aside the factual case, which stands on its own. You so ably demonstrated that point this morning in your factual presentation.

On page 4 of your testimony, the last two sentences of the first paragraph, are you saying that epidemiological studies linking a certain disease to toxic chemical exposure is all that is necessary in a court of law to determine culpability in toxic substance personal injury litigation cases?

Mr. **Milford**. Yes, I am saying that, Congressman. The epidemiological studies, human studies, are the best evidence available on the cause and effect relationship between chemical and disease. There is no better evidence.

The only other available avenue is to simply wait until all the bodies drop and count the bodies. I think that that has been demonstrated to be an inappropriate scientific measure. That is the type of evidence that is used—that combined with animal studies.

I should add that in many cases, one epidemiological study, if it is a valid epidemiological study that shows a connection between exposure and disease, is more than sufficient in many toxic substances cases, certainly to go to the jury.

Mr. DASCHLE. So what you are saying is that the VA is going beyond what any court of law would require to establish that relationship?

Mr. MILFORD. That is correct. What the VA is doing is now ignoring epidemiological studies that show a cause-and-effect relationship, and if I understand the Administrator's testimony this morning and statements made also by other scientific advisers to the VA, they would have this policy decided on the basis of counting epidemiological studies.

Now, the public health policy of this Federal Government has been for years that an epidemiological study is conclusive proof of a cause-and-effect relationship.

We don't wait until the epidemiological studies add up. We don't wait for years and years and years. We act on the basis of the best available evidence.

Mr. DASCHLE. We talked about double standards just a few minutes ago and I am amazed that that stark a double standard exists in current benefit provisions.

On page 11 of your statement on the cardiovascular presumption, I am wondering how many studies did it take?

It is my understanding that there was just one study on cardiovascular disease that you cited. Were there any other citings that caused the VA to come to the conclusion to increase the disability benefits?

Mr. MILFORD. In this case, there is only one mandated epidemiological study. Only one study was done that served as the basis for the presumption.

Mr. DASCHLE. So in that case, they did go by the court of law in a sense? The court of law says there only has to be one, and in this case they used only one and came up with the conclusion that disability compensation could be increased.

Mr. MILFORD. That is right.

Mr. DASCHLE. You have done the committee a real service, and I want to thank you, Mr. Milford, for an excellent statement.

Mr. MILFORD. Thank you.

Mr. APPLEGATE. Thank you, Mr. Daschle.

Does counsel have any questions?

Mr. WILSON. Mr. McEwen has asked that sometime during the hearing, if possible, that we hear from other witnesses, Dr. Franz Ensinger, who is the director of the soft tissue tumor section of the Armed Forces Institute of Pathology; a Dr. Donald Tschudy, a senior investigator in the metabolism branch of the National Cancer Institute and a representative from the American Medical Association, Council on Scientific Affairs.

Mr. Chairman, if I just may say something about this circulatory matter that Mr. Daschle has referred to and that the witnesses have referred to, that has quite a substantial history in the Veterans' Administration.

It was involved with the Disabled American Veterans making a rather involved study of that issue. Later on that study was brought to the attention of the Congress and the Veterans' Administration that it might be wise to ask the Veterans' Administration and the DAV to comment upon that.

Mr. APPLEGATE. Thank you very much.

Mr. MOON. In response to Mr. McEwen's request, Dr. Ensinger and Dr. Tschudy will be contacted. In fact, one of them has been contacted already, and will be testifying probably in June on the issue of soft tissue sarcoma.

Mr. APPLEGATE. Thank you, again, Mr. Milford. We appreciate your testimony.

Mr. MILFORD. Thank you.

Mr. APPLEGATE. I would add at this point that Mr. Montgomery, our chairman, had to leave for another meeting, and he has asked for unanimous consent that his statement be inserted in the hearing record. Without objection, that will be done.

### STATEMENT OF HON. G. V. "SONNY" MONTGOMERY, CHAIRMAN, COMMITTEE ON VETERANS' AFFAIRS

Mr. MONTGOMERY. Mr. Chairman, at this point in the hearing I would like to comment on the testimony we have heard so far concerning this important issue. We have been told by several witnesses that they are basing their views on soft-tissue sarcomas on the Swedish studies, and we just heard Mr. Milford tell us the Swedish studies would stand up in a court of law. I would like to introduce into the record a letter from our Past Chairman Ray Roberts to the National Academy of Sciences requesting their evaluation of the Swedish and German sarcoma studies and President Philip Handler's response that includes his staff commentary which states, and I quote, "The five papers represent very little substantive data about the association and even less about the causation of cancer by the phenoxy herbicides and their contaminants." This June 1980 position was reaffirmed yesterday by the staff.

Additionally, I would like to place in the record a paper by Doctor Lawrence Hobson of the VA's agent orange project office, dated March 1983, that lists the clincial work concerning soft-tissue sarcoma association with phenoxy exposure which lists at least 10 other countries' studies that do not support the soft-tissue sarcoma association.

And lastly, I would like also to place in the record an extract of a paper from the American Council on Science and Health that contains their position statement quoted in part, "No scientific reports presented to date have shown any convincing relationship between the traditional use of 2,4,5-T and adverse health effects on humans."

I recognize that the N.A.S. position of the Swedish studies was made almost three years ago, and I suggest, Mr. Chairman, that you invite them to testify at the hearing you plan to conduct later this summer as I think it is very important to our deliberations on this bill we are considering today.

MAY 30, 1980.

Mr. PHILIP HANDLER,
*Chairman, National Research Council, National Academy of Sciences, Washington, D.C.*

DEAR MR. HANDLER: The Committee on Veterans' Affairs recently received the enclosed five scientific papers on chemical exposure in Sweden and Germany.

In order that we may add to our knowledge about the Agent Orange issue, I would appreciate you advising me at your earliest convenience of the consensus opinion of the National Research Council concerning the methodology, findings and conclusions of these papers.

Sincerely,

RAY ROBERTS, *Chairman.*

Enclosure.

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NATIONAL ACADEMY OF SCIENCES,
*June 26, 1980.*

Hon. RAY ROBERTS,
*Chairman, Committee on Veterans' Affairs, U.S. House of Representatives, Washington, D.C.*

DEAR MR. CHAIRMAN: This will acknowledge your letter of May 30, transmitting for comment five scientific papers on exposure to chemicals in Sweden and Germany. While we have no active study under way concerned with these particular chemical substances, the staff of our Assembly of Life Sciences have reviewed the papers accompanying your request and their comments are incorporated in my response. This staff commentary should not be considered an official Academy statement, since our limited resources and the desire to provide a prompt response would not permit the in-depth analysis and other processes that would normally accompany studies undertaken by the Academy on behalf of the government.

From their vantage point, our staff believe that the five papers represent very little substantive data about the association—and even less about causation—of cancer by the phenoxy herbicides and their contaminants. Staff conclusions concerning the five studies are based primarily on the methodologic weaknesses that characterize the performance of these studies as reported, associated primarily with an imprecise assessment of exposures (recall methods are in most cases greatly flawed) and with the inability to dissect the contribution of any chemical substance over that of the totality of exposure from occupational and life—style characteristics. From these studies, one can perhaps conclude an association between occupation and cancer but certainly no attribution can be made with exposures to any particular chemical. Other difficulties arise from the enrichment of the study population by selecting cancer patients and from the choice of cancer types for which there are no known causes.

These types of data are not uncommon and in our experience best serve as stimuli to broad-gauge investigations whose design favors the generation of data sufficiently sensitive to discriminate among various chemicals in the environment. (However, we also recognize that at times such refinements are beyond the ability of our science.) The advancement of our knowledge and the magnitudes of proclems may now warrant a national commitment for such an epidemiologic undertaking with regard to the pheonxy herbicides and to TCDD or "dioxin."

If the Academy can be of further assistance, please do not hesitate to call upon us.

Sincerely yours,

PHILIP HANDLER, *President.*

# EPIDEMIOLOGY OF SOFT-TISSUE SARCOMA AND RELATED HUMAN RESEARCH

(as related to herbicide exposure)

LAWRENCE B. HOBSON, M.D., Ph.D.
Deputy Director
Agent Orange Projects Office
Veterans Administration
March 1983

## Work Supporting Soft-Tissue Sarcoma Association with Phenoxy Exposure

| Author & Year of Report | Location | Study Type | Result | Conclusion |
|---|---|---|---|---|
| Hardell 1977 [2-3] | Sweden | Clinical observation | 7 cases of soft-tissue sarcomas in foresters | Suggests need for study. |
| Hardell & Sandstrom 1978,1979 [3-4] | Northern Sweden | Case-control of foresters & wood workers exposed to phenoxy acids and/or chlorophenols | Risk of soft-tissue sarcomas increased 5.3 times | "An increased risk for soft-tissue sarcomas" but "a specific evaluation of the effect of separate chemical substances was not possible." |
| Eriksson, Hardell et al 1979, 1981 [4-5] | Southern Sweden | Case-control chiefly of farmers | Risk increased 6.8 times | "Exposure to phenoxy acids and chlorophenols might constitute a risk factor in the development of soft-tissue sarcomas." |
| Honchar & Halperin; Zack & Suskind; Cook; Moses & Selikoff; Johnson et al (Hardell & Eriksson agreed) 1980-1981 [8-9] | U.S.A. Monsanto and Dow | Piecemeal observations; number of exposed persons not given | Total of 7 soft-tissue sarcomas of varied types | Results "suggest a common pattern" and "fit in with" Swedish results. |
| Sarma & Jacobs 1981 [9] | U.S.A. VAMC | Clinical observations | 3 cases of soft-tissue sarcoma of varied types in Vietnam vets | Isolated observations without basis for interpretation. |
| Barthel 1981 [9] | East Germany | Cohort of 1,791 pesticide sprayers | One "soft-tissue malignant tumor" | No comment on single soft-tissue malignancy. Bronchogenic carcinoma most common malignancy. |

Work Not Supporting Soft-Tissue Sarcoma Association

| Author & Year of Report | Location | Study Type | Result | Conclusion |
|---|---|---|---|---|
| Riikimaki 1982 (Reeky-mocky) [9-10] cide users | Finland | Cohort of 1,926 pesti-exposures" and cancer risk. | No soft-tissue sarcoma | "Results do not confirm the association between mixed herbicide. |
| Smith et al 1982 [10-11] | New Zealand | Case-control of phenoxy users | No increased risk (odds ratio 1.6) | Results "do not generally support the hypothesis that exposure to phenoxy acid herbicides cause soft-tissue sarcoma." |
| Hogstedt & Westerlund 1980 [10] | Sweden | Cohort of 386 foresters | No soft-tissue sarcoma | Risk of cancer increased. |
| [illegible]g & Granstam 1979 [11] | Sweden | Cohort of 375 foresters | No soft-tissue sarcoma | Fewer deaths from cancer than expected; greater risk of renal & lymphatic-hematopoetic cancers. |
| Jirasek (also Pazclerova-Vejlupkova) 1981 [11] | Czechoslavokia | Cohort of 55 heavily exposed workers | No soft-tissue sarcoma | Bronchogenic carcinoma in 2 workers. |
| May 1982 [11] | Coalite Co., Gr. Britain | Cohort of 79 exposed chemical workers | No soft-tissue sarcoma | No cases of malignant neoplasm. |
| Thiess & Frentzel-Beyme 1982 [11-12] | BASF, West Germany | Cohort of 74 exposed chemical workers | No soft-tissue sarcoma | Increased risk of cancer, especially gastric carcinoma. |
| Huff et al 1980 [11] | Philips-Duphar, Holland | Cohort of 93 exposed chemical workers | No soft-tissue sarcoma | No cancer cases in 14 years. |

Work Not Supporting Soft-Tissue Sarcoma Association

| Author & Year of Report | Location | Study Type | Result | Conclusion |
|---|---|---|---|---|
| Caramachi et al 1981 [12] | ICMESA, Seveso, Italy | Clinical observations of 5400 exposed persons | No soft-tissue sarcoma reported (after 6 years) | No unusual pathology reported. |
| Young et al 1978 [12] | Other industrial accidents, e.g. W. Germany, USSR, France, USA, AUSTRIA | Observation for various periods | No soft-tissue sarcoma reported | Poorly controlled observations. |
| AMA Council 1981 [15] | U.S.A. | Unsystematic observation | No increase in soft-tissue sarcomas noted | After more than 30 years of widespread phenoxy herbicide use, no convincing evidence that they cause any cancer in man. |

## References Critical of Soft-tissue Sarcoma Work

| | |
|---|---|
| Remington (OTA Consultant) 1980 | Swedish "results are consistant with the hypothesis that phenoxy acid exposure increases the risk of tumors of this type" but the "case control methodology is intrinsically susceptible to subtle and unmeasurable biases." |
| Jannerfeldt 1980 | Questionnaire and interview techniques introduce and statistical methods are questionably appropriate. |
| Coggan & Acheson 1982 | Same objections plus finding many different tumors (Including Lymphatic malignancies and gastric carcinoma) with exposure weakens case. Also no comparison was made of types of soft-tissue sarcomas in cases and in controls. "It is as yet impossible to estimate with any precision the risk of soft-tissue sarcoma due to phenoxy herbicides." |
| _Lancet_ editorial 1982 | Supports Coggan and Acheson |
| AMA Council 1981 | "While 2,4,5-T and 2,4-D have been used in agriculture, forest management and residential landscaping for over 30 years, there is still no convincing evidence that they and/or TCDD are -- carcinogenic -- in man." |

NOTE:

Hardell, Axelson & colleagues have written some 6 papers in refutation of the criticism. Hardell [6-7] did case-control study of carcinoma of colon that showed no relation to phenoxy herbicide exposure, offered as evidence of lack of bias in questionnaire-interview technique. However, Hardell used as control data from other studies.

Other Studies Cited with Soft-tissue Sarcoma Research

| Author & Year of Report | Location | Study Type | Result | Conclusion |
|---|---|---|---|---|
| Malignant Lynphomas | | | | |
| Hardell 1979 [5] | Sweden | Clinical observation | 14 of 17 patients reported phenoxy or chlorophenol exposure | Suggests need for study. |
| Hardell, Eriksson et al 1980, 1981 | Northern Sweden | Case-control | Risk increased 6 times with phenoxy herbicides but 8.4 for chlorophenols & 11 times for solvents & phenoxy herbicides | Results suggest "that exposure to organic solvents, chlorophenols, and/or phenoxy acids constitutes a risk factor for malignant lymphoma," including Hodgkin's disease. |
| Axelson & Sundell 1974, 1977, 1980 [1-2] | Sweden | Cohort of 348 railroad workers (208 exposed to phenoxy herbicides) | One "Hodgkin's lymphoma" | See under "Carcinomas". |
| Bishop & Jones 1981 | England | Clinical observation | One non-Hodgkin's lymphoma among 158 workers | Exposure was to pentachlorophenol. |
| Barthel 1981 [9] | East Germany | Cohort of 1,791 pesticide sprayers | One lymphosarcoma, one "malignancy of lymphoid tissue" | Chemical to which exposed is uncertain. |
| Carcinomas | | | | |
| Axelson, Sundell et al 1974, 1977, 1980 [1-2] | Sweden | (1) Cohort of 348 railroad workers<br>(2) Case-control study of cancer | Increased risk of death, especially from amitrol, but also from phenoxy herbicide exposure | Increase was slight, greatest for gastric carcinoma after use of phenoxy herbicides. |

| | | | | |
|---|---|---|---|---|
| Thiess & Frentzel-Beyne 1982 [11-12] | BASF, West Germany | Cohort of 74 exposed chemical workers | Increased risk of cancer but lower overall death rate | Increase was less than 2 fold for cancer, greatest for gastric cancer. |
| Uirasek 1981 [11] | Czechoslovakia | Cohort of 55 heavily exposed chemical workers | Two deaths from cancer | Both deaths were bronchogenic carcinoma. |

# THE HEALTH EFFECTS OF HERBICIDE 2,4,5-T

A Report by the American Council
on Science and Health

The American Council on Science and Health is an independent educational association promoting scientifically balanced evaluations of chemicals, the environment and human health.

The Council is a nonprofit association exempt from federal income tax under Section 501 (c)(3) of the Internal Revenue Code. All contributions are tax deductible as provided by law.

Individual copies of this report are available at a cost of $10.00. Prices for ten or more copies are available on request.

This report on 2,4,5 - T was researched and written by Mary Katherine Hayes, M.S., Research Associate for the American Council on Science and Health (ACSH).

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## POSITION STATEMENT

Based on its review of the scientific evidence, the American Council on Science and Health (ACSH) concludes that there is insufficient evidence to support a ban on 2,4,5-T. No scientific reports presented to date have shown any convincing relationship between the traditional use of 2,4,5-T and adverse health effects in humans.

The toxicity of 2,4,5-T and its contaminant, 2,3,7,8-TCDD (2,3,7,8-tetrachlorodibenzo-p-dioxin), has been demonstrated under laboratory conditions at doses far higher that those to which humans are exposed. Estimates of human health risk derived from animal experiments are unreliable. Furthermore, those risks which have been calculated for 2,4,5-T and 2,3,7,8-TCDD are extremely small.

ACSH recommends that the current uses of 2,4,5-T in rice fields and rangeland be continued and the suspended uses in forests, railways and highways, and landscaping be reinstated.

ACSH recognizes the problems posed by 2,4,5-T's unavoidable contaminant, 2,3,7,8-TCDD, and urges that every effort be made to further reduce contamination during the manufacturing process. ACSH recommends that applications of 2,4,5-T, particularly aerial spraying, be strictly monitored to minimize unnecessary environmental and human exposure. ACSH believes that stringent safeguards for the manufacture and application of 2,4,5-T will effectively reduce any potential adverse effect of 2,3,7,8-TCDD while allowing for the continued use of the herbicide.

Mr. **Daschle**. Mr. Chairman, could I ask unanimous consent, these two guests of the VFW could come before the committee very briefly. Commander Currieo was here earlier and made reference to them.

They are in the audience and at the end of our witness list, if they could simply come for just a few minutes to be able to express themselves, even if the Chair would have to be vacated, I would be happy to take over, to allow them to testify today.

As I understand it, they have come from quite a distance. I would be very appreciative.

Mr. **Applegate**. Thank you, Mr. Daschle. I was going to honor your earlier request that they be recognized. When we are through, I would be happy to do so.

Next we have Lt. Col. David Passamaneck, USA, Retired, national legislative director of the AMVETS. Colonel Passamaneck, it is good to see you again.

## STATEMENT OF LT. COL. DAVID J. PASSAMANECK, U.S. ARMY (RET.), NATIONAL LEGISLATIVE DIRECTOR, AMVETS

Colonel **Passamaneck**. Good afternoon, Mr. Chairman.

Mr. Chairman, gentlemen, the American Veterans of World War II, Korea, and Vietnam appreciate the opportunity to present our views concerning the highly sensitive issue of the infectious consequences of the exposure of our service personnel in Southeast Asia during the period of the Vietnam war to phenoxy herbicides, including so-called agent orange.

Since the issue was raised by concerned Vietnam veterans approximately 15 years ago, we have witnessed a continous monumental exercise in the generation of documents reflecting the collective lack of definitive medical and scientific information regarding the epidemiological connection between exposure to agent orange and various physical disabilities, including cancer, skin lesions, birth defects, liver disease, and even psychological problems.

This documentary mountain has been generously contributed to by the Veterans' Administration, the Congress, other Government agencies, and many private citizen groups, including, of course, veterans organizations.

Two substantive legislative enactments have resulted from the widespread concern about the subject. Public Law 96-151, signed on December 20, 1979, directed the VA to conduct an epidemiological study of persons who were exposed to phenoxy herbicides and to determine the medical consequences, if any, of that exposure.

Interagency contracts have been signed between the VA and the Centers for Disease Control and expenditure of approximately $3 million has been approved by the Office of Management and Budget to conduct the study which optimistically speaking should result in a first report to Congress sometime in 1988 or 1989.

In addition, Public Law 97-72 offers Vietnam veterans who request it priority physical examinations and diagnostic studies which could result in prolonged treatment for those conditions not medically determined to be caused by anything other than agent orange.

We appreciate the motivational thrust of the Congress in positively highlighting the problem in Public Law 97-72.

Without reiterating the sad and often confusing story of delay and inaction by the VA in the conduct of the epidemiological study mandated by Public Law 96-151, we would simply endorse the remarks of the former Administrator of Veterans Affairs, Mr. Nimmo, in August 1982, when he said, "I found our research stuck on dead center. People had good intentions, but nothing was happening."

We are sorry to say that Mr. Nimmo's remarks are still applicable at the present time.

AMVETS views the purpose of H.R. 1961 as building a much needed fire under the VA to spur completion of the determinative studies relating to agent orange with a view toward ending the terrible uncertainty and anxiety afflicting our Vietnam veterans and their families, and compensating those who may be found entitled to service-connected benefits as the result of exposure to this lingering hazard of their service.

We believe, however, that the responsibility for creating epidemiological presumptions in title 38 belongs to the Congress and should not be shifted to the VA or any other executive agency.

It is one thing to build a fire under the VA to hopefully insure compliance with Public Law 96-151 before all of our affected Vietnam veterans become too old and sick to take advantage of it. It is quite another thing to require the Administrator to gather the firewood and supply the matches.

AMVETS, therefore, supports enactment of H.R. 1961 with the exception of that portion of section 3 which adds subsection (d)(2)(A)(IV) and (B) to section 312 of title 38, United States Code.

That is the section that authorizes the Administrator to add other presumptive conditions to the list which would be covered by the same situation relating to agent orange.

This concludes my testimony, gentlemen. I am prepared to answer any questions if you have them.

[The prepared statement of Colonel Passamaneck appears on p. 323.]

Mr. **Applegate**. Thank you, Colonel Passamaneck, for appearing before our committee. Yours was a very eloquent statement. We appreciate it, and it will certainly be useful in our deliberations.

The gentleman from South Dakota, Mr. Daschle.

Mr. **Daschle**. I just concur in your comments, Mr. Chairman.

Colonel, I tell you, you could read a telephone book and make it sound eloquent. You have a way about your presentation that I wish a lot of us had. You really put some fire into what you say and you speak with a tremendous authority.

Just on the way you said it, I don't think any of us dare disagree. But I commend you. It is an eloquent statement, a strong statement, and we are grateful to have that kind of support for this legislation. We appreciate it very much.

Colonel **Passamaneck**. We appreciate what you have been doing, sir, in a decidedly lonely battle to get some justice in this situation.

Mr. **Daschle**. Thanks to you and a lot of others, it is not nearly as lonely as it once was. Thank you.

Colonel **Passamaneck**. Thank you, sir.

Mr. APPLEGATE. What you are saying is that is the way we ought to say it when we are out on the political stump.

Mr. DASCHLE. I don't care who knows it.

Mr. APPLEGATE. Thank you, Colonel Passamaneck.

Colonel PASSAMANECK. Thank you, sir.

Mr. APPLEGATE. Our next witnesses are Mr. John Terzano, legislative director of the Vietnam Veterans of America, accompanied by Mr. Leslie Platt, special counsel.

### STATEMENTS OF JOHN F. TERZANO, LEGISLATIVE DIRECTOR, VIETNAM VETERANS OF AMERICA, ACCOMPANIED BY LESLIE A. PLATT, SPECIAL COUNSEL

Mr. TERZANO. Thank you, Mr. Chairman. I know we are under some tremendous time constraints today, but I would like to hit some of the highlights of my written statement and then allow Mr. Platt to address some of the comments that were made here earlier today.

Mr. Chairman, and members of the subcommittee, I am John Terzano, legislative director of the Vietnam Veterans of America. With me here today is Mr. Leslie Platt, VVA special counsel.

Four years ago, the Vietnam Veterans of America was the first witness at the first congressional hearings on agent orange, and the possible adverse health effects it may have caused to those who served.

In those hearings, VVA called for the establishment of a presumption of service connection for cancer and liver disorders experienced by America's Vietnam veterans.

Those hearings in 1979 culminated in the introduction in the Congress of the first bill specifically focused on the problems of agent orange. That bill was introduced by Congressmen Thomas Daschle and David Bonior, who were then and remain today pioneers and leaders in the fight for basic equity for those who served their country in Vietnam.

Much has happened since those first hearings. Perhaps most important, official Washington at both ends of Pennsylvania Avenue now recognize that Vietnam veterans may already be or may in the future become ill as a result of their service in Vietnam.

This recognition was slow in coming and much yet remains to be done. But for those of us who have been at the forefront of this fight for official recognition of an attention to our concerns, for those of us who were ridiculed, scorned and in some cases thought to be just plain off our rockers in the early stages, for those of us who have seen our views recognized in Federal law and multimillion-dollar Government research budgets, for us the progress of the recent past is only the beginning.

Our concerns are now shared by millions of Americans who are becoming acutely aware of the potential hazards of these chemicals. But what we seek, Mr. Chairman, is basic equity for Vietnam veterans while we wait for the scientists to finish their end of the job.

That is why VVA is here today, approximately 4 years after those first hearings and 5 years after agent orange first surfaced as a national issue, again presenting testimony on behalf of the Vietnam veterans in this country on the same core issue, presumption of service connection for disability compensation.

Mr. Chairman, I am not here today to detail the sound, scientific basis for H.R. 1961. There is no doubt that the time has long since come for the Congress to provide such presumptions.

In a different world, the Veterans' Administration itself might well have done so under its administrative authority, but as with priority health care for Vietnam veterans, congressional directions by statute were necessary.

We asked several questions about the issue of compensation. How can the same Federal Government that buys out Times Beach, Mo., for over $30 million continue to turn its back on a group of individuals who have given so much of themselves for their country.

What kind and how much scientific data will be enough? When will the Congress, the executive branch and the Veterans' Administration acknowledge and deal responsibly with the existing data which were deemed sufficient for addressing Times Beach, but are somehow found deficient for America's veterans?

Also, after years of denying chloracne claims, the VA now insists that it pays those claims. But what was that one piece of additional evidence that finally tipped the scales of justice toward the veteran and what piece of evidence will convince the VA, again, to tip the scales in favor of the veteran?

Mr. Chairman, H.R. 1961 is limited legislation. It is moderate and carefully circumscribed. If it becomes law, it would only help a small number of sick veterans.

Yet, VVA believes it is an important step as it will finally establish a statutory framework for dealing responsibly with the compensation issue by placing the Government on the side of the veteran.

VVA submits that there is now enough evidence on which a Congress can and should act. We submit that the Congress need only look at the VA's own scientific literature review and EPA's recent actions to see that evidence.

Yet, there are those who say that the evidence is not enough. There are those who feel that we still need to wait.

It has been said on numerous occasions that we cannot afford to become complacent because a comprehensive research agenda is being carried out.

Even the best effort of which our scientists are capable may not ultimately provide the definitive and uncontrovertible scientific information about the health effects of phenoxy herbicides.

Jones E. Bernstein, the chair of the original interagency work group on phenoxy herbicides, stated in testimony over 3 years ago that, in short, "we may be left after the research is done with many of the same social policy issues we face today."

Mr. Chairman, the time has come for Congress to have the courage to begin to confront the compensation issues. H.R. 1961 is a good bill, and it should be passed now.

VVA strongly supports this legislation. The care and effort that Mr. Daschle and his staff and many other members have put into developing this legislation is clearly evident.

H.R. 1961 is a bare minimum that the Government can and should do. In fact, the bill, in my opinion, does not go far enough.

Volume I, chapter 5 of the VA's literature review lists a number of diseases for which there is ample scientific evidence to support presumptive compensation eligibility for additional disabilities.

Specifically, the VVA would strongly recommend making the following changes:

A broader liver disorder presumption to include, but not be limited to, chronic hepatitis and other liver disorders whose etiology is other than congenital or alcohol related. Not only is there evidence in the literature review to support this, but just last week the New York Times reported that 112 out of 130 residents of Imperial, Mo., whose blood and urine were tested by the Centers for Disease Control were told that they had abnormalities in their samples.

A broader skin disorder presumption. Thousands of veterans have complained to the VA about various skin problems. While the VA claims that only a few are chloracne and that it does treat and compensate them, it does nothing for the overwhelming majority of those who have other skin problems.

A presumption of peripheral neuritis, which is a loss of sensory function or numbness.

Finally, a presumption for lymphomas.

In closing, Mr. Chairman, the agent orange story is one in which the basic issue is the Government's integrity and credibility. This committee has the opportunity to strengthen that integrity and credibility by moving forward with this compensation legislation.

Then when the final chapter is written on the treatment of Vietnam veterans, this committee will not be remembered among those who told America's Vietnam veterans that they, and the sacrifices they made over a decade ago, have been forgotten.

The committee's action on this legislation will be noted and remembered.

Mr. Chairman, H.R. 1961 is now the litmus test by which Vietnam veterans across the country gage not only this committee, but also this Congress commitment to Vietnam veterans.

Thank you.

[The statement of Mr. Terzano appears on p. 327.]

Mr. **Applegate**. Thank you very much, Mr. Terzano.

You state, I think it is on page 2, at the top, that you seek basic equity for Vietnam veterans while you wait for the scientists to finish their job.

Ultimately what you are saying is that presumption of service connection for disability compensation for serious health problems may be attributable to exposure to herbicides. Do you have questions in your mind?

Are you saying that these health problems may be attributable to exposure to phenoxy herbicides?

Mr. **Terzano**. I feel that they are attributable to exposure to phenoxy herbicides. Mr. Platt?

Mr. **Platt**. If I might elaborate a little bit. I think the Vietnam Veterans of America believes the fundamental issue is one of the relative risk faced by those who served in Vietnam of incurring adverse health effects of a serious nature as a result of that service, and the proofs on which assertions of an increased risk are based with respect to exposure to phenoxy herbicides, the literature, the scientific community is replete with studies that are creditable, that demonstrate the increase in risk following either acute or chronic exposure.

Indeed, the VA's worldwide literature review itself is replete with such examples. If I might ask to cite to you and have entered into the record the entire document prepared under the auspices and by contract for the Environmental Protection Agency of the U.S. Government, in 1980, prepared a scientific report called Dioxins.

In the introduction to the health effects, section 6 of that document, there is a paragraph that reads as follows:

> On a molecular basis, 2,3,7,8-TCDD is perhaps the most poisonous synthetic chemical. As shown in Table 35, only bacterial exotoxins are more potent poisons. Not only is this TCDD isomer extremely poisonous, but it also has extremely high potential for producing adverse effects under conditions of chronic exposure.
>
> Human exposure to 2,3,7,8-TCDD has induced chloracne [an often disfiguring and persistent dermatologic disorder], polyneuropathy [multiple lesions of peripheral nerves], nystagmus [involuntary rapid movement of the eyeball], and liver dysfunction as manifested by hepatomegaly [increase in liver size] and enzyme elevations.
>
> In animals, this compound has been shown to be teratogenic, embryotoxic, carcinogenic, and cocarcinogenic.
>
> It has been established that under certain conditions 2,3,7,8-TCDD can enter the human body from a 2,4,5-T-treated food chain and can accumulate in the fatty tissues and secretions.
>
> The available data indicate significant risks associated with the use of dioxin-contaminated herbicides.

There is no doubt, Mr. Chairman, that in the view of the people who prepared this report under contract for the Environmental Protection Agency in 1980, which was reviewed, as indicated, in the preamble by a number of representatives of the chemical industry and other people knowledgeable in the field on the scientific literature available at that point that there was in the literature at that point enough on which to make the statements that are the consensus views of those who prepared the report, though not necessarily agreed to by all of the commentators in that report.

Subsequently, the Veterans' Administration in preparing its worldwide literature review under Public Law 96-151 documented a number of studies which indicate that either acute or chronic exposure to phenoxy herbicides can increase the human health risk or in animal studies has been shown to result in an increase in the risk of disorder in the animal.

If I might just complete the answer then, I guess the point is that the "may" and the "shall" are dealt with in a complementary fashion by saying that there is significant evidence, substantial evidence on the record now that demonstrates that there is, indeed, an increased risk from exposure to phenoxy herbicides.

The question of the degree to which that increased risk possibility may be manifest in an individual veteran who was on the ground in Vietnam will not probably ever be known with respect to each and every individual veteran.

However, the studies that are now being planned or underway will tell us whether as a group ground troops who served in Vietnam are running an increased risk of disease as a result of those exposures.

Mr. APPLEGATE. Thank you very much, Mr. Platt. Does that mean that you want to keep the statement as it is?

Mr. PLATT. If I might add an additional item.

Mr. APPLEGATE. In other words, do you want to keep it as "may" rather than "shall" or "is"?

Mr. PLATT. I think that the "may" in the preamble that says, indeed, that with respect to an individual veteran who served in Vietnam and who is now sick, his illness may indeed be the result of exposure to herbicides or other environmental factors in Vietnam is appropriate.

I also think that it is appropriate based on the scientific evidence to state in the legislation, that is, the substantive part of the legislation, that there shall be a presumption of service connection.

Indeed, for purposes of exposure, the VA policy already is, as I understand it, that anyone who was in Vietnam is conceded to have been exposed to phenoxy herbicides.

Mr. APPLEGATE. Thank you for your explanation.

The gentleman from South Dakota.

Mr. DASCHLE. Mr. Platt, you certainly have enlightened this committee, I think, a great deal with regard to many of the studies, especially the EPA study.

It seems to me that what you are saying is that regardless of the Swedish studies, regardless of the Australian studies, there is enough documented information within our own country to justify passage of this legislation based on the relationship that these studies have shown; is that your understanding?

Mr. PLATT. Yes, sir, I would, if I might, in further response refer to an article that appeared in the New York Times in January of this year, and I would like to quote briefly from that, in which it says:

> In regard to the concern about the possibility of adverse health effects from exposure just to dioxin, the possible risk posed by even that level of dioxin must be weighed, industry spokesmen said, against the enormous economic value of the products.
>
> Industry specialists say they believe, further, that the risk is not so high as to require special precautions other than those already taken in handling hazardous substances like herbicides and pesticides.
>
> Assessing that risk, of course, is part of the problem. Researchers say the risk begins to appear in animals with exposure in the parts per one thousand billion range. The Centers for Disease Control have advised Missouri officials that materials containing more than one part per billion of TCDD should be removed from residential areas, but that figure itself has caused some confusion.
>
> Dr. Renado de Kimbro of the Federal Disease Agency expressed annoyance with some news accounts that reported incorrectly that the agency considered dioxin concentrations below one part per billion in the soil to be, quote, unquote, safe.
>
> Although this is the detection limit being used in soil tests of residential areas in Missouri, she said, it represents a compromise between health concerns and cleanup practicalities.
>
> Dr. Kimbro said exposure to lower concentrations over extended periods could still present health risks.

The article goes on, again, to demonstrate the views of the Centers for Disease Control who have testified numerous times before this committee about their concerns in this regard.

There is no doubt that there are many questions yet unanswered. There are probably some questions that never will be answered with respect to individual veterans.

However, in answer to your question, there is also no doubt that there is a credible body of scientific evidence that now demonstrates that there is significantly increased risk to health from exposure to phenoxy herbicides.

Mr. DASCHLE. I would say that from what you have read that it goes way beyond just an amount of credible evidence. It seems to me it is a compelling amount of evidence. From the information you provided the committee this morning, I think that we can make that case even clearer.

It is disappointing to me to see some come before this committee and say that in spite of all this overwhelming evidence, they would have our veterans wait another 10 or 15 years.

I don't understand that line of thinking. But let me ask you another question with regard to the Australian study. You have done some work on that, Mr. Terzano, Mr. Platt. Is it your understanding that the study dealt with birth defects and offspring or did it go anywhere beyond that?

Mr. TERZANO. Mr. Daschle, I find it interesting that when we have an overabundance of credible scientific evidence, everybody wants to dismiss it. But when you get a report which is all the Australian study was, which was a report on birth defects and infants, they have not completed a full-scale epidemiological study; they have not completed a mortality study or morbidity study.

In fact, their courts have even directed their equivalent of our Veterans' Administration not to allow any more claims to be brought up in front of their courts. For example, in what has become known as the *Collins Simpson* case, here was a guy who had a lymphoma who tried to get compensation from their veterans administration.

They denied it. He subsequently died and his widow took it to their court system. The courts ruled in favor of the widow and instructed their VA that they didn't want to be burdened with these type of cases and their VA should begin to adjudicate these claims the right way.

You know, maybe that is what we need here in this country is a little bit of judicial review on this agent orange issue. But it is interesting to me that whenever we have something that is what I call a false negative, which the Ranch Hand is going to come up, of course, with some evidence that is going to say that basically, probably, the Ranch Handers are going to be OK; that people who are opposed to this type of legislation are willing to hang their hat on just this one small piece of puzzle.

To me, it doesn't jive.

Mr. DASCHLE. Mr. Terzano, I certainly sympathize with your feelings. I get that same kind of an impression from time to time. Perhaps that is what this hearing will bring out, that there are studies and ample evidence already on the books which currently show that there is a tremendous increase in the hazard to your health by exposure to dioxin and you have lended additional credibility to that debate and the argument.

We certainly are grateful to you for presenting your testimony this morning.

Mr. APPLEGATE. Thank you, Mr. Terzano, and Mr. Platt, for appearing before the committee. We appreciate your testimony.

Mr. **Applegate.** Mr. Daschle, would you like to introduce your two new witnesses.

Mr. **Daschle.** Unfortunately, I don't know their names, but Don Schwab is here, a representative of the VFW, if he could so identify them, I would be grateful to him.

Mr. **Schwab.** Thank you, Mr. Chairman.

The commander in chief brought in to witness these hearings two witnesses, each accompanied by an attendant. The first one is Billy Housewright from Tennessee, accompanied by his wife; and the second one is Delmont Murphy from Portland, Maine, who is accompanied by Mr. Penwit.

Mr. **Applegate.** Thank you. Would you gentlemen please come to the witness table? Gentlemen, we appreciate your appearing before the committee. If you have something that you would like to say, we would like to hear it.

## STATEMENT OF BILLY HOUSEWRIGHT, VIETNAM VETERAN

Mr. **Housewright.** My name is Billy Housewright, and I was in the U.S. Army in Phu Bai. That is above Da Nang.

I was in the outfit—the young man was talking about—the 505. That is part of I Corps. That is what I was in. I have never met anybody before that was in that, so I would like to talk to him.

Anyway, the 505, which I was in in 1970—was something sort of like an infantry outfit. We rode on gun trucks, that sort of thing. So I guess he was before my time and we progressed on up.

Anyway, I was assigned to an outfit that hauled jet fuel back and forth across the country. Since I was infantry, I couldn't do truck driving, so I was left out on the perimeter guards and this kind of thing.

I noticed regular sprayings, C-130's. That was in 1970. I never thought much about it because I know the Government is not going to do anything to hurt you or anything like that. So I never said anything about it.

I got a kind of a nervous disability. I figured it was caused from agent orange. Almost total body hair loss. The only hair I got is on my head.

Right before I went to Vietnam that is what I looked like. When I come back, it all came out in clumps. I have rashes which are red and turn brown, and older rashes which are brown that turn to black.

I am slow to feeling pain. This is an outline I made up because I have sort of a memory loss.

I have sudden aches and pains. My biggest worry is what might be going on inside of me, what will I be like in a few years.

I met him last night [indicating] and he was telling me that I was progressing along like he used to be. I was hoping to meet other Vietnam veterans.

I have been going to the VA hospital in Tennessee, Johnson City, called Mountain Home. I started in 1972, when all my hair was out. I got out of the Army in 1971; February.

They gave me a large box of medicine. I made three trips. The third trip they told me not to come back because they done all they could do for me.

In 1980, I returned for agent orange test, which is a blood test, and I had a chest X-ray, which I got. My blood test came back. Thought I had a kidney problem, a liver defect of some sort. They got that through my blood test.

But nothing was ever said about it. So I made three trips recently, which my wife put me in the paper because we were having serious marital problems. I get violent at times, and sudden bursts of anger and that sort of thing; nightmares.

So she put my picture in the paper and got me back to the VA hospital, which I refused to go, and people came and talked to me.

I have had a lot of trouble. My last appointment was last Thursday, the 21st. I finally got to see a skin specialist then. It was his first day on the job. His name was Shermer.

He said I need to see a liver specialist. He is going to do some biopsy on me if the VA will let him. I talked to Dr. Fulton over at the VA hospital and he said they don't do skin testing biopsy.

Anyway, Mr. Shermer put in my medical records that I should get a liver specialist. When he took my records back where they are supposed to go to, while I was standing there to get a slip to get some ointment for my head, this woman called and they tore out the part of my record that says I should see a liver specialist.

I guess they could read the rest of these. I am kind of excited. I am looking for treatment. So far I have not got a whole lot.

My friends are being kind to help me. They put some things together like Indiana, New Jersey. I guess my closing statement would be for my own part I am not looking for money, just some help medicalwise.

Mr. **Applegate**. Thank you very much.

Mr. Murphy, do you have a statement for the committee?

## STATEMENT OF DELMONT MURPHY, VIETNAM VETERAN

Mr. **Murphy**. Yes. I am also a veteran of Vietnam. I am a retiree from the Air Force. I spent 20 years and 18 days. Ten months after I retired I came down with this problem.

I was sent to a neurologist and was diagnosed as having what they call spasmodic claudicolus.

I later went to the VA hospital in Maine. They didn't know what I had. After three visits there, they finally sent me to Jamaica Plains VA Hospital in Boston. I spent a total of 6 months down there.

They diagnosed me as having dystonia muscalorem diforum, which in layman's terms is a disease of the central nervous system. I asked them how this was caused. Did they have any idea how this was caused? They said, "no, we don't know." I said, "is there any cure?" They said, "no, there isn't that we know of, not at this time."

I also went through this agent orange exam. In fact, recently. In doing the agent orange exam, you talked with the doctor. As my friend here was saying, you go through, they give you X-rays, urinanalysis and blood tests and so on.

The particular doctor that I talked to at the VA hospital, he asked me, he says, "What do you know about agent orange?"

I said, "I know quite a bit about it. I read brochures. I have read different articles, different magazines and newspaper stories and so on."

When I got done telling the doctor what I knew about agent orange, he sat there with pen in hand with my medical records open and said, "I just don't know what to write." He said, "You know more about it than I do."

This is a doctor with the VA. So I guess I had to just leave it up to him to what he wanted to write down. Evidently, he should have let me write it down because I knew about it.

I have heard a lot of testimony here today. I have heard a lot of questions come up about exposure to agent orange. Everybody in Vietnam was exposed to agent orange. This was in a book put out by the VA.

Some areas heavier than others. For instance, Nha Trang, I think Quang Tri Province, Da Nang, Toung Song Nu, places like that were heavily exposed. Other areas, all areas, everybody in Vietnam was exposed, either heavily or lightly.

I heard testimony—heard somebody mention something about a glass of water. I just was speaking to a friend of mine back a while ago, and I said I would like to take a glass of this water and take an aerosol can with dioxin in it and spray it into the water and offer anybody in this room to see if they would drink it.

I don't say that sarcastically. I say this honestly. I don't think anybody in this room would drink that water.

Like I say, I have heard testimony from the DAV, and I am a lifetime member of the DAV. I am a member of the American Legion. I am a lifetime member of the VFW. I have heard all this testimony today.

Some I concur with. Some I do not concur with. I, myself, personally believe that I was exposed to agent orange. I know I was exposed.

Like I say, they can't explain to me what this is. You mention agent orange at the Veterans' Administration at the hospital, they don't want to talk about it. They say, well, he will either walk out or we will see you later.

I didn't have a prepared statement because in order to read, I have to lay down to read. I can't get my head down far enough to read.

So what I am saying, I am just saying from my personal feelings and from the way I believe, and I would be glad to answer any questions from any member of the committee.

Mr. APPLEGATE. Gentlemen, thank you very much. I would say that both of you expressed yourself exceedingly well. I think you got your points across.

I know it is particularly painful to come before a congressional committee or to publicly talk about the ills or disorders that you have.

I know that we have a particularly difficult chore in front of us both in receiving all of the testimonies and then trying to reach a conclusion regarding the question of service connection.

If this turns out to be the case, then we are going to proceed with either this legislation or some other legislation that is going to provide that those who served in Vietnam, like other veterans before them, receive that which they have earned.

I, for one, appreciate what you two, as well as other veterans of the Vietnam conflict and all veterans of all our Nation's wars have done to preserve and to protect and defend the Constitution of this country which allows us to sit in this body in the first place and to be able to listen and try to do something to help our veterans.

I don't think there is really any other country in the world that can make that claim. I think that is why there have been so many people that have been willing to go to defend our Nation. We talk about our freedoms in this country, but freedom is not free and it is not cheap. It is indeed very expensive.

It comes at the cost of so many lives. I think all of us who serve on this committee are very aware of that. We do appreciate what you have done and we are going to see that you get what you fully have earned and deserve.

Mr. MURPHY. I thank you, gentlemen. I thank you for listening to us. We really appreciate the opportunity to come here and to speak and to say what we think and what we believe.

Mr. APPLEGATE. Thank you. The gentleman from South Dakota, Mr. Daschle.

Mr. DASCHLE. Mr. Chairman, I fully concur with your eloquent remarks.

I started out the hearing today by making a comment that seems to be very apropos as we end this particular session. We can be quite detached from all of this unless we take the time to learn of these diseases and what they do.

Many are dreadful. They destroy the body. They have to represent the closest thing to hell that we can find here on Earth. When you look at a victim's agonizing eyes and listen to his story, you change.

You lose the detachment and you develop a deep commitment to find out how we can help these people. Your presentation, I think, has given us cause to have that deep commitment. We are very grateful to you for your statements and for the help that you have presented today in your testimony.

Thank you all very much.

Mr. APPLEGATE. That concludes the hearing for the subcommittee for today. I remind you that we will meet tomorrow morning at 9 a.m. in this room.

Thank you for being here.

[Whereupon, at 12:50 p.m., the subcommittee was recessed, to reconvene at 9 a.m., Wednesday, April 27, 1983.]

# H.R. 1961, VIETNAM VETERANS AGENT ORANGE RELIEF ACT

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## WEDNESDAY, APRIL 27, 1983

House of Representatives,
Committee on Veterans' Affairs,
Subcommittee on Compensation, Pension and Insurance,
*Washington, D.C.*

The subcommittee met, pursuant to notice, at 9:10 a.m., in room 334, Cannon House Office Building, Hon. Douglas Applegate (chairman of the subcommittee) presiding.

Present: Representatives Applegate, Daschle, Montgomery, and McEwen.

Also present: Representative Edgar.

### OPENING STATEMENT OF CHAIRMAN APPLEGATE

Mr. Applegate. The subcommittee will come to order.

This morning we will continue our hearings on H.R. 1961, the Vietnam Veterans Agent Orange Relief Act.

Yesterday we heard from various veterans organizations. Today we will hear from several professional witnesses from the private sector and from the VA. Miss Dorothy Starbuck, Chief Benefits Director of the Veterans' Administration, will be the principal witness for the Veterans' Administration.

At this time I would like to recognize my good friend and colleague from Ohio, Mr. McEwen.

Mr. McEwen. Thank you, Mr. Chairman.

I would reiterate my expressions of respect and appreciation for holding these hearings that I voiced yesterday on this vitally important bill, not only to Vietnam veterans but to all veterans and all of us who are concerned about veterans.

I wish to take this moment, also, Mr. Chairman, to point out, as you are aware, that we are shortly beginning markup in the Economic Development Subcommittee of the Public Works Committee on a bill that is of vital importance to you and to myself and those of us from Ohio. It is only because of the confidence I have in your leadership that I will have to excuse myself shortly. But I want to emphasize again my respect for the sponsor of this legislation, my support for it, and my concern that this be given a complete and full hearing and action by our committee.

Thank you, Mr. Chairman.

Mr. Applegate. To start off this morning we will first hear from the Honorable Barbara Boxer from California. Barbara, I want to welcome you to the subcommittee. Your interest and concern for our Nation's veterans is appreciated very much.

Your complete statement will be entered into the record and you may proceed in any manner that you wish.

## STATEMENT OF HON. BARBARA BOXER, A REPRESENTATIVE IN CONGRESS FROM THE STATE OF CALIFORNIA

Ms. BOXER. Thank you very much, Mr. Chairman, for this opportunity to appear before you.

I am very moved by this entire issue. I don't know if you have heard a recent song about Vietnam by Billy Joel which says, in part, "We came in spastic—we left in plastic—as numbered corpses". For many of those who didn't come home in a box, for the many of those whose names are not listed on the Vietnam Memorial, life has become a tortured and difficult existence. And the VA's painfully slow decisionmaking on the health aspects of agent orange is making it more tortured and more difficult.

Despite numerous respected studies linking the diseases in H.R. 1961 with agent orange, there is still not automatic compensation to Vietnam veterans suffering from certain cancers and skin ailments.

What can these Vietnam veterans do to make the Federal Government listen? Under the Feres doctrine, emanating from a Supreme Court decision, they cannot sue the Federal Government for service-related ailments, although their exposure to agent orange is unquestioned. And the decision to use agent orange was made by the U.S. Government. Lawsuits against the chemical companies which produce agent orange are a slow and arduous procedure.

The answer is, Mr. Chairman, that we in the Congress are their only hope. They can't sue. The VA hasn't been moved despite the studies. The veterans in my district tell me that since the VA rejects agent orange claims as a matter of course, many veterans don't even bother consulting the Veterans' Administration.

Mr. Chairman, I want to give you three very brief histories of veterans in my district. Due to the abbreviated time I have before you, these capsule histories do not even begin to convey the scope of the suffering endured as a result of exposure to agent orange and other herbicides used in Southeast Asia.

Case No. 1, a veteran in Vietnam from 1967 to 1969 as an Army infantry medic. He saw helicopters spraying defoliants and he camped in and traveled through defoliated areas, drinking the water from the ground and the rivers. He has suffered from—and this is a partial list—a brain tumor, which was operated on, skin rashes and spots, liver disease, stomach disorders, headaches, dizziness, insomnia, depression, rages, decreased smell and taste, stiff and numb joints and extremities. He has a son born with birth defects—deformed toes on both feet—who has difficulty in walking, who gets sick often. The veteran applied for VA benefits and received a letter from the VA conceding his exposure to herbicides in the service, but denying compensation.

Case No. 2, a veteran in Vietnam from 1968 to 1969 serving as a Marine Corps infantry squad leader. He saw defoliants being sprayed, passed through defoliated brush, walked, slept, drank from, and bathed in defoliated areas. He has suffered from testicular cancer which required surgery, and which has resulted in impotency and sterility. Other medical problems include chest pains, night sweats, abdominal cramps, skin rashes with blisters, stiffness, pain, numbness in extremities, and personality changes. He filed a VA compensation claim which was denied.

Case No. 3 died at age 35 of carcinoma of the lungs. He was in the Marine Corps for 10 years and served in Vietnam from March to September 1968. He slept, worked, and walked through defoliated areas sprayed with herbicides. The VA, Mr. Chairman, denied any claim that his illness was a result of agent orange exposure. He left a widow and three children aged 3, 5, and 11.

Mr. Chairman, we don't know how to undo the physical damage caused by agent orange, but we will try. We don't know how to undo the psychological trauma of the Vietnam war, but we will try. But what we can and must do now is compensate all of those who have been wounded in our wars.

If someone lost an arm or a leg in service to our country, he or she was rightly compensated. If someone lost sight or hearing, he or she was rightly compensated.

The chemicals in agent orange inflict a different kind of damage—slower, sometimes more subtle damage, but without question, damage, sometimes fatal damage. This damage must be compensated. We have the studies. We have been patient.

The Congress sent men and women to Vietnam to fight. Now the Congress must be ready to pick up the pieces. The pieces are there in my California district, and all over this Nation, where several thousand men and women continue to seek help.

I commend Mr. Daschle for authoring H.R. 1961. I commend your committee for holding these hearings, and I am very proud to be supporting this bill.

Again, thank you, Mr. Chairman.

Mr. APPLEGATE. Thank you very much for a very compassionate, articulate and eloquent statement. Your concern for the Vietnam veteran is very obvious and we certainly appreciate your presentation before the committee.

The gentleman from Ohio.

Mr. MCEWEN. Mr. Chairman, I have no questions, except to express my appreciation as well for her testimony and obvious concern about this important issue.

If I could just take a moment of the committee's time in a different context, I believe that this member has done us a service by bringing to our attention a song by Mr. Billy Joel, whoever that is, in which he mentions that——

Ms. BOXER. I think he has better name ID than I do.

Mr. MCEWEN. Well, I can assure you, he doesn't with this member, because anyone who refers to those who fought for freedom at the call and service of their country, and died for the effort as "numbered corpses," is not a reflection of how we on this committee view those who have served our country and those who have made the supreme sacrifice.

Now, maybe they were just numbers to certain rock singers during that time maybe they still are to this day. But as the member pointed out very appropriately, the Congress sent these men and women to Vietnam to fight, and as we see what happened in the wake of that service and the genocide that swept through Southeast Asia with the administration of Pol Pot and others, I believe that that sacrifice should not be diminished by references to them as though they were mere numbers. They were human beings and they were important people that met the call of their country, right or wrong, as the administration at that time chose. We can all look back with 20-20 hindsight, but I can assure you that the members of this committee respect that service, admire that service, and will do what we feel we can do to compensate that service.

I say again, for the benefit of this one member, that those rock singers that stood there and made life miserable for those who were attempting to serve their country, to now turn around with statements of this type, that I have only contempt for them.

Thank you, Mr. Chairman.

Ms. BOXER. Mr. Chairman, if I might respond to my good colleague.

First of all, I would like to send you the entire song that I quoted from because it is nothing of the sort that you're making it. It is a very compassionate statement. It is not a political statement. It is not an argument about whether the war was right or wrong, and that is not why I am here, either. I think what the song does, it humanizes the people that were caught in that war, for whatever reason. I would be very happy to send you this because it is not the intent of the song to demean anyone. On the other hand, it is to call attention to the problems.

Mr. APPLEGATE. I would like to recognize the gentleman from Pennsylvania, Mr. Edgar who, though while he is not a member of the subcommittee, is sitting in. Mr. Edgar is chairman of the Hospital and Health Care Subcommittee which has just come back from a very important field trip, holding hearings on our veterans hospitals.

Mr. EDGAR. Thank you, Mr. Chairman.

Thank you for your statement. When I was listening to your conversation about the music, I thought about James Watt's statement a couple of weeks ago, where he became a music critic. I tend to feel that as public officials maybe we ought to listen to the words and not the critics of the kind of music that is presented.

There appears in your statement, and I think generally, that there is substantial evidence to proceed on a limited basis with the presumption of these three disabilities that are included in Tom Daschle's bill. Where there is disagreement or question, undoubtedly there will always be those kinds of disagreements. That does not mean that we should not respond to the problem when no one else will, and you made that statement in your comments.

In this Congress alone there are currently 10 pieces of legislation dealing with presumption for agent orange disabilities, and there have been 14 congressional hearings on the subject since 1980 alone. Both the American people and the Vietnam veterans have come to Congress to express the alarm about the potential effects of dioxin exposure, and the issue has been raised thoroughly in the press, by veterans organizations, and by the scientific community.

The Federal Government is spending millions on studies. We study everything. We seek answers where there might not be any answers in many of these areas. Our credibility is at stake, I think, as Members of Congress, and you as a new Member of Congress have done well to come and make a very clear statement.

In other countries, such as Australia, Vietnam veterans have been able to go to the courts and resolve their grievances. In our democracy, unbelievably, veterans are not allowed to file their claims through the courts. In that regard, veterans are second-class Americans.

In lieu of that, we are the judges and the jury here in Congress. In that instance, when no one else can make up their minds, when there appears to be no scientific consensus, then I think there must be a political concensus.

Let me quote the Chief Medical Director, Dr. Donald Custis, in testimony before the Subcommittee on Oversight and Investigations last September. This is September 15, 1982, just 8 months ago. He said at the time:

> It may well be that Congress cannot wait for scientific answers in the short term, in which case it may well be that the sociopolitical aspects of this problem will have to be addressed.

This committee has approved legislation in the past granting presumption for disability compensation for medical conditions with a lot less information and proof than we have before us today concerning agent orange. In doing so, we did what Dr. Custis said, we addressed the sociopolitical aspects of this problem.

I think—and I hope my colleagues agree—that we ought to support Mr. Daschle's effort and that we must be the judges, we must be the jury, and that the Vietnam veterans have waited very long and I think they await our decision on this action in this year. I have been on this committee for 8½ years and we began to address the problem. I think a number of people began to talk about it, and I think in the limited way that Congressman Daschle's bill is designed, we can address that presumption and move on to hopefully getting some more information from the studies.

Mr. Chairman, thank you for letting me take the time, and I commend you for your statement.

Ms. **Boxer**. Thank you very much.

Mr. **Applegate**. Thank you, Mr. Edgar. Thank you, Ms. Boxer, for——

Mr. **Daschle**. Mr. Chairman, could I——

Mr. **Applegate**. I'm sorry, Tom. I didn't see you come in.

The gentleman from South Dakota.

Mr. **Daschle**. Barbara, I had a chance to read your statement. I'm sorry I wasn't here for the oral presentation. But I want to thank you very much for a very eloquent statement. You are a fan, as I am, of that song by Billy Joel. I think it tells a lot. I had thought at one time of just having the words of that song be my testimony, because——

Mr. **McEwen**. I object. [Laughter.]

Mr. **Daschle** [continuing]. Because it is a tremendous song, in spite of the critic on my right.

I want to thank you very much for your testimony.

Mr. **Edgar**. Would the gentleman yield? Is that critic on your far right or just to your right?

Mr. **Daschle**. Moderate right. [Laughter.]

Ms. **Boxer**. Mr. Chairman, if I might, I also had thought of not speaking and just playing the song. I'm kind of glad I didn't choose that because it might not have gone over too well.

In any case, you weren't here and I really do want to commend you for doing this for all of us and for the Vietnam veterans.

Mr. **Applegate**. Thank you, Barbara.

Ms. **Boxer**. Thank you, Mr. Chairman.

[The prepared statement of Hon. Barbara Boxer appears on p. 331.]

Mr. **Applegate**. Our next witness is Dr. Samuel Epstein, Department of Preventive Medicine, University of Illinois Medical Center, Chicago. Dr. Epstein, welcome to the committee.

Your statement, Doctor, will be inserted into the record. You may summarize, if you wish. I would remind everyone that we are under the 10-minute rule, and the 5-minute rule for questions.

## STATEMENT OF DR. SAMUEL S. EPSTEIN, PROFESSOR OF OCCUPATIONAL AND ENVIRONMENTAL MEDICINE, DEPARTMENT OF PREVENTIVE MEDICINE, UNIVERSITY OF ILLINOIS MEDICAL CENTER, CHICAGO, ILL.

Dr. **Epstein**. Mr. Applegate, members of the subcommittee, it is my privilege to testify before you this morning on H.R. 1961.

Briefly, I am an M.D., a pathologist, and toxicologist, who has specialized in problems of toxic chemicals in the environment with particular relation to delayed chronic effects, including cancer, birth defects, and genetic abnormalities. I have about 250 scientific publications in this area, besides half-a-dozen books.

Apart from my scientific interest in these matters, I have consulted to Congress, including the Senate Committee on Public Works, for several years. I have been a consultant and expert witness to several Federal agencies. I chaired two panels on Secretary Finch's 1969 Commission on Pesticides and Their Relationship to Environmental Health—the panels on birth defects and on genetic abnormalities, the former of which unanimously recommended that the use of phenoxy herbicides such as 2,4,5-T should be restricted to prevent human exposure.

It was also my privilege to testify in 1970 before Senator Hart on the hazards of 2,4,5-T and, more recently, in 1980, on H.R. 6377 before the Subcommittee on Medical Facilities and Benefits. I have also been actively involved in problems of dioxin contamination in Missouri and have testified before the Missouri legislature.

I would like to commend Congressmen Daschle and Applegate on H.R. 1961, which I regard as a limited step in the right direction of redressing major inequities in compensation.

I would first of all like to discuss briefly problems in the scientific demonstration of causality. The level of scientific proof generally attainable and required for the demonstration of causality is conventionally defined in terms of presumptions or probabilities, rather than more absolutely. However, for agent orange disease, not only is a high or absolute level of proof inappropriately demanded by the Veterans' Administration, but also the burden of proof is inappropriately shifted to the veteran.

The demonstration of causality in chronic disease due to toxic exposures requires evidence of exposure and of adverse effects, and evidence of positive associations between exposure and such effects based on toxicological and/or epidemiological studies.

Evidence relating exposure and adverse effect is based on statistical grounds of probabilities and not absolutely. For Congress to demand absolute proof is to misunderstand the whole scientific basis of demonstrating causality.

As far as animal evidence of adverse effects is concerned, it is clear that dioxin is about 200,000 times more acutely toxic than cyanide. In addition, we are dealing with an agent that induces cumulative toxic effects in animals. Therefore, results of continual exposures accumulate and add up.

One of the characteristics in animal studies of dioxin effect are the delayed chronic effects. Another characteristic is the multisystem disease. Dioxin is unique insofar that it produces diseases in a wide range of organ systems including liver, kidney, central nervous system, endocrine system, immunological effects, metabolic effects, effects on blood lipids and effects on porphyrins. In some species, dioxin produces chloracne and in other species it doesn't. In the species in which it doesn't produce chloracne, you still get evidence of dioxin effects. The suggestion that you have to have chloracne to have evidence of dioxin effects is inconsistent with an overwhelming body of scientific data.

Over and above the multiorgan effects, dioxin produces a wide range of reproductive effects—birth defects, toxicities of the growing embryo, gonadotoxic effects, sterility, and infertility, and it does these at levels as low as part per trillion. It is difficult to comprehend the fact that we are dealing with agents so powerful that the toxicity is not measured as we usually do, in parts per thousand or parts per million or parts per billion, but in parts per trillion.

Additionally, dioxin has been shown to be carcinogenic in a wide range of animal studies, in many species, producing cancers down to the lowest levels tested of five parts per trillion; the cancers involve a wide range of organs, including soft tissue sarcomas.

Additionally, there is an overwhelming body of regulatory precedent in the use of animal data as a valid basis for action designed to prevent human exposure. There are numerous such precedents in the Environmental Protection Agency, the Occupational Safety and Health Administration and the Food and Drug Administration. In other words, when you have data from valid, well-designed animal tests, there is an overwhelming presumption that such data are extrapolatable to the human situation. There is also an overwhelming consensus in the international scientific literature that once you have data from valid, well-designed animal tests, these data can be extrapolated to the human situation.

The World Health Organization, the International Agency for Research on Cancer, recognizes dioxin as a potential human carcinogen. To suggest, therefore, that the animal data represents some extraordinary aberration of limited relevance to the human situation is, to my mind, an example of special pleading and disregarding of science for the sake of special interests.

The human evidence on toxicity of dioxin comes from a wide range of sources, quite apart from the limited studies on Vietnam veterans, including studies on occupational accidents—and there have been about 10 such studies; studies on occupational exposures from handling contaminated intermediates such as trichlorophenol; occupational exposures during agricultural, forestry, or rights-of-way application of herbicides; community exposures from dioxin-contaminated waste oils, such as in Times Beach, and presumed community exposures in Midland County, Mich., in the vicinity to the Dow Chemical facility, whose soil is heavily contaminated with dioxin. I stress, that the symptomatology and effects induced by dioxin reflect a wide range of factors including dosage or exposure levels, duration of exposure, presence of other toxic chemicals and contaminants, and age, sex, and reproductive status of exposed populations.

Before discussing the human data further, let me just point out that there is a very substantial consistency between the effects demonstrated and observed in clinical and epidemiological studies in humans and those described in a very wide range of animal studies. There appears to be considerable misunderstanding as to the role of chloracne in agent orange disease, with particular regard to its alleged obligatory or pathognomonic value as an index of exposure. It has been suggested that, if you don't have chloracne, you don't have dioxin poisoning. This is scientifically untenable. As I pointed out already, there is marked species specificity in experimental animals. In a wide range of experimental animals, you can get chronic dioxin toxic effects, including cancer, in the absence of any chloracne.

Chloracne also has protean manifestations in humans ranging from the classic chronic pustular cystic form to much milder and less characteristic forms with minimal residual scarring, hyperpigmentation, and depigmentation. The chloracne is generally, but not invariably, associated with a wide range of other symptoms and objective signs of multisystem disease.

Now, there are numerous reports in the literature of a wide range of toxic effects of dioxin in humans, including metabolic abnormalities, abnormalities in phospyrins and lipids, sometimes associated with athrosclerosis. There is also a wide range of case studies of soft tissue sarcoma observed in humans, particularly in workers following exposure to dioxin, and in the absence of preceding chloracne. Therefore, to suggest that unless you have chloracne you don't have dioxin disease is to deliberably misunderstand or misread a vast body of scientific data to the contrary. Chloracne is certainly not an obligate determinant of dioxin toxicity.

As far as multisystem disease is concerned, dioxin is well recognized in the clinical and toxicological literature as a potent multisystem toxic agent producing a wide range of acute and also delayed effects, many of which can progress to the chronic. Recognized symptoms include weakness, pain in limbs and joints, insomnia, photosensitivity, nausea, vomiting and diarrhea. Recognized signs include abnormalities in liver function, clinical or subclinical peripheral neuropathy, elevated blood lipids, psychological and personality disturbances, and also cardiovascular problems.

This disease complex is generally consistent with that recognized in preliminary surveys of Vietnam veterans and with those recognized following occupational accidents.

Now, as far as cancer is concerned, I would point out that there have been several series of studies on the carcinogenic effects of dioxin, both in Sweden and elsewhere, and in general, these studies have demonstrated a consistency of effects.

First of all, in Nitro, W. Va., we have seen soft tissue sarcomas in Monsanto workers, besides an excess of lymphomas and leukemias. In a series of Swedish occupational studies, we have seen soft tissue sarcomas, besides other cancers such as malignant lymphoma and lung cancer. In the United States, there have been a series of accidents and explosions in chlorophenol plants, and the National Institute of Occupational Safety and Health, in a series of pooled cohort studies, has demonstrated a fortyfold excess of soft tissue sarcomas in occupationally exposed workers, including Dow and Monsanto workers. We have seen a major excess of soft tissue sarcomas in women living in the vicinity of the Dow plant in Midland, Mich. Finally we have also seen soft tissue sarcomas in Vietnam veterans.

As far as reproductive effects are concerned, there have been many studies on birth defects following exposure to phenoxy herbicides all over the world. But, in addition, I would like to very briefly mention recent findings in Midland, Mich. which have demonstrated not only an excess of birth defects, but also cleft lip and cleft palate in the early seventies, and also a marked decrease in fertility rates of wives of dioxin-exposed workers in the Dow facility.

Additionally, I would point out that some of these effects are consistent with those described and recognized in Vietnam veterans, including problems of infertility.

I would briefly like to comment on H.R. 1961. H.R. 1961 establishes a presumption of causality between agent orange exposure and three categories of adverse health effects—soft tissue sarcomas, porphyria, and chloracne, and for other unspecified disease conditions as determined by the discretion of the VA Administrator. This restriction to the specified disease categories is, however, at variance with an extensive body of toxicological, clinical, and epidemiological data on the effects of dioxin and, as such, improperly excludes a wide range of dioxin-induced diseases. The discretion afforded the Administrator to recognize and compensate other dioxin-induced diseases appears to reflect an unduly optimistic faith in the agency's ability and interest to take such steps.

An alternative concept which this subcommittee may wish to consider is the establishment of a nonrebuttable presumption of causality for the specified diseases and also for those other diseases which are consistent with the scientific literature on dioxin toxicity. Such presumptions would be still further strengthened by the absence of other defined and exclusive etiologies for the disease in question.

For agent orange disease, not only is a high level of absolute proof inappropriately demanded by the VA, but also the burden of proof is shifted to the veteran. While H.R. 1961 is a commendable step in the direction of reducing this heavy burden, it fails to reflect the major inconsistencies with established practices in compensating other diseases such as multiple sclerosis, diabetes and cardiovascular disease in an amputee.

The VA compensates a veteran if he develops multiple sclerosis within 7 years of leaving the military. Where is the evidence on the relationship of multiple sclerosis and service-connected disability? Where is the evidence on cardiovascular disease following amputation, for which the VA compensates, with the exception of one single study quoted by the National Academy of Science Committee, which simply demonstrates a statistical excess, without scientific evidence on causality. Here, when it comes to agent orange disease, we are apparently prepared to discount statistical evidence; but in multiple sclerosis, we are prepared to accept the relationship for compensation in the absence of any evidence whatsoever, as far as cardiovascular disease, similarly.

Such inconsistencies raise to my mind the suspicion that constraints to the recognition of the extent of agent orange disease and the need for its equitable compensation may reflect political and economic considerations rather than truly scientific considerations. Such constraints are often manifest in recommendations to postpone further consideration of compensation pending results of further studies in the future. Such constraints are also in striking contrast to a wide range of Federal acts of restitution in instances when data on causality are much less well developed, such as compensation for the Times Beach buyout.

Illustrative of such problems of such demands for future study is the Ranch Hand study which has been well and truly criticized by a wide range of groups and expert bodies, including the National Academy of Science. Among other things, we must appreciate that even if the Ranch Hand study were to have scientific merit—and this is debatable—it will take another 20 or 30 years before the results of this study become available.

Additionally, the subcommittee may wish to consider the inconsistencies between the heavy burden of proof still imposed on the Vietnam veterans with recent Federal action on Times Beach.

Finally, the Congress should consider the belated need to shift the burden of compensation for agent orange disease from the U.S. taxpayer to Dow Chemical. Dow Chemical publicly asserted in 1964 that 2,4,5-T was absolutely nontoxic to humans or animals. However, Dow Chemical then had specific information on the presence and high toxicity of the dioxin contaminant. It deliberately suppressed this information from the USDA and from the Department of Defense, and as a result of this, very many young U.S. citizens were unnecessarily exposed to this highly toxic contaminant. It is difficult to understand why the U.S. taxpayer should shoulder this burden of restitution pay for acts of omission and commission by Dow Chemical and other industries in this regard.

Thank you, sir.

[The statement of Dr. Samuel Epstein appears at p. 334.]

Mr. APPLEGATE. Thank you very much, Dr. Epstein. We appreciate your very concise and very technical piece of testimony.

I notice in your testimony you had mentioned the term "Agent Orange disease." This is something that we have referred to many times but I have never heard it used by anyone in the medical community. Is this a term that is now accepted and used today in the medical community?

Dr. EPSTEIN. I think it has become recognized in the last 4 or 5 years that dioxin produces a very wide range of diseases, and as the extent and the scope of these diseases has become more and more apparent, as the understanding of soft tissue sarcomas and other cancers besides the wide range of metabolic and organ diseases has become recognized. It has thus become appropriate to use the term "Agent Orange disease" as a shortcut to embrace this wide array of different toxic effects induced by Agent Orange.

This also recognizes the fact that dioxin produces effects in a wide range of systems. In other words, it is a multi-system toxic agent and there is overwhelming toxicological data, besides clinical and epidemiological data, in this respect.

Mr. APPLEGATE. Thank you, Doctor.

Mr. Bonior mentioned yesterday that he would like to subpena the Dow Chemical records, and he mentioned in particular the Midland Dow wives' birth defects study. It was stated by our chairman that there was a copy of that study and it was placed in the record.[1] He had made a quote from the study, that "it is our interpretation that under the conditions of this study, there were no biologically meaningful associations between the various pregnancy outcomes and paternal dioxin exposure." I would like for you to comment on that.

Also, on page 16, it mentions that:

> There is growing and substantial evidence of a clear consistency between the wide range of toxic effects induced by dioxin in experimental animals of various species and those observed in a wide range of exposed human populations, including occupational and Vietnam veterans. This consistency relates to multi-system disease, reproductive toxicity * * *

I would like to hear just a little bit more about that and what your feelings are with regard to the Australian study.

Dr. EPSTEIN. First of all, let me comment that the documentation which Dow Chemical has made available is not responsive to the subpena. There is a wide range of aspects of the reproductive effects which have not been included. For instance, in the study on the Dow wives, originally when the study was designed, there were three groups. One, the wives of dioxin-exposed workers; two, the wives of Dow workers who were not exposed to dioxin; and three, wives of workers outside of the Midland, Mich., facility.

It is very interesting that data on the third group have never been made available. In other words, we really don't have a negative control for these studies. What we have are wives of Dow workers, all of whom have had varying degrees of dioxin exposure—clearly, the wives of the dioxin-exposed workers have had maximal exposure—but the wives of the non-dioxin workers in Dow have also had exposure.

Second, we do not have data on all the years of the study. We only have limited data which have been made available. But even of the limited data which has been made available, let me give you the following figures which demonstrate that there are paternally-mediated effects.

There were 371 wives of dioxin workers; there were 737 conceptions among these 371 wives. There were 345 nondioxin Dow wives, and there were 2,031 conceptions in this group. In other words, the wives of Dow workers had a threefold decrease in conception rates. This is consistent with the effects of dioxin in animals, producing infertility, quite apart from sterility, and also inconsistent with the data—sporadic and poorly systematized though it may be—on the Vietnam veterans on the problems of infertility and hyperfertility.

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[1] See p. 25.

Mr. McEWEN. Dr. Epstein, could you elaborate? I mean, are there other factors involved there? The age of the employees, was that a more senior position or were there any other variables that should be considered, or are the rest of them pretty——

Dr. EPSTEIN. I beg your pardon. I have already indicated that there are limitations in these studies insofar that the true negative controls have been omitted for some reason, and Dow has never explained the reasons why it has omitted the data on the wives of the nondioxin workers at Midland.

However, Dow's own data demonstrates that, in their own comparisons of wives of dioxin workers with the wives of workers who weren't handling dioxin in the facility, that there was a threefold decrease in conception rate in their own study when they matched the workers. That is my point. And this is also consistent with toxicological effects in animals. It is consistent with the railroad studies which a U.S. jury recently made an award of $58 million for railroad workers who were exposed to dioxin. It is consistent with a wide range of other reports, quite apart from the reports on Vietnam veterans.

Additionally, there are many other pieces of information which Dow has refused to make available, including information as to how it has discharged and how it has disposed of its dioxins in the last 20 years. I should remind you—although it isn't of critical interest to this committee—that the facility at the Dow Midland, Mich., plant contains soil levels up to 100 parts per billion, levels of dioxin contamination very close to those of the Times Beach area.

Mr. APPLEGATE. Doctor, in the interest of time, I would like to move on to some of my colleagues. I had asked about the reproductive toxicity with relationship to the Vietnam veteran and your thoughts on the Australian report. What I am going to do is submit that to you, and if you would, I would appreciate it if you would further address those for us.

Dr. EPSTEIN. With pleasure.

Mr. APPLEGATE. At this time, I recognize the gentleman from Ohio.

Mr. McEWEN. Thank you, Mr. Chairman.

Dr. Epstein, it is always a pleasure to have you come here, and I greatly enjoyed your testimony. I would like to ask, for my own benefit, an additional question.

In recently reading about the reports to HHS and HEW concerning saccharine and all that, in the medical journal there was a great deal of criticism as to the reserved use of certain animals and relating those findings to human. I notice on page 7 of your testimony, you mention the consideration of chloracne, that mice and rats being resistant, and rabbits and monkeys being sensitive.

Do you have any insights, as an expert in this field, that would be helpful to our committee in this area?

Dr. Epstein. Yes. I think, first, I should point out that the whole basis of modern science, of the pharmaceutical industry, modern medicine, is the validity and extrapolation of data from animal tests, from properly, well-designed valid animal tests to the human situation.

Now, animals, like humans, are not homogeneous. In any one strain, in any one species, you may find individual variations, and particularly as far as toxic effects are concerned, there very often are variations in strain effects from animal to animal.

However, what is remarkable about dioxin is its consistency in the toxic effects in a wide range of body systems—liver, kidney, nervous systems—irrespective of strain and species.

Now, as far as skin effects are concerned, there is certainly a variation in sensitivity on animals, depending on what kind of skin they may have and other factors which have been poorly determined. Clearly, we do know that in sensitive species, like monkeys, the effects of dioxin producing chloracne depend on dose. At relatively high doses you get chloracne; at very much lower doses you don't get chloracne, but you can get other chronic toxic effects.

As far as cancer is concerned, there again is a remarkable consistency irrespective of whether you test mice, rats, or monkeys, because they all get cancer. The fact is this: In the independent scientific community, there is an overwhelming consistency of opinion and acceptance of the fact that information derived from valid world-designed animal tests can be extrapolated to the human situation.

There has been some misunderstanding and problems in the carcinogenicity test because clearly you can't take 30 animals in a test and test these animals with a chemical of the same level of exposure as massive human populations are exposed to and expect to be able to pick up a 1 in 10,000 or 1 in 1,000 cancer effect because you only deal with 30 animals. Therefore, in an attempt to reduce the gross insensitivity of animal tests as a function of sample size, you test at higher doses than those of human exposure.

But, as far as dioxin is concerned, we see a wide range of toxic effects in animals at levels much below those of human exposure. Don't forget, when we are talking about the Vietnam veterans, we are dealing with people whose exposure was to dioxin at levels going as high as 70 parts per million. In Times Beach, where you had dioxin in soil, you were dealing there with 100–200 parts per billion, twenty-fold lower levels.

Mr. McEwen. Thank you.

Before the green light goes out there, I have one additional question.

You mentioned in your testimony that the discretion afforded the Administrator of the Veterans' Administration in this bill to recognize and compensate other dioxin-induced diseases appears to reflect an unduly optimistic faith in the agency's ability and interest to take such steps.

Would you elaborate on that for us, please?

Dr. Epstein. Perhaps that was somewhat ad hominem, but I think if you examine the past record of the Veterans' Administration, you will find it isn't characterized by exactly a well-developed understanding of problems of agent orange toxicity or a desire to compensate victims for agent orange disease. The fact that Congress has seen fit to remove the authority for a wide range of studies from the Veterans Administration to other organizations is perhaps an additional point in this direction.

Mr. McEWEN. Thank you very much.

Thank you, Mr. Chairman.

Mr. APPLEGATE. The gentleman from South Dakota and the prime sponsor of H.R. 1961, Mr. Daschle.

Mr. DASCHLE. Thank you, Mr. Chairman.

Dr. Epstein, we are very grateful to you for the expert testimony you have provided us again on the scientific aspects of this issue.

I have a couple of questions that really were addressed in your testimony but I think need elaboration. At the very end of your oral presentation you made reference to Ranch Hand. You mentioned that there is a difference of opinion within the scientific community on the value of Ranch Hand.

Could you elaborate on that a little bit?

Dr. EPSTEIN. I really wasn't aware of a difference of opinion on the subject. I was under the impression that there was an overwhelming consensus that the Ranch Hand study was poorly designed, suffered from major defects, had low levels of sensitivity, and in addition to these problems, the fact that even if the Ranch Hand study is prolonged for a 20- or 30-year observation period, it is going to take that period of time before one can possibly arrive at definitive conclusions.

Mr. DASCHLE. Are you talking 20 or 30 years for the carcinogenic effects?

Dr. EPSTEIN. For long-term chronic effects with particular reference to carcinogenicity, yes.

Over and above that, there are very serious questions as to the relevance of the Ranch Hand study to ground troop exposure. The patterns of exposure in the ranch hand study were totally different to those of ground troops. In the Ranch Hand study, Air Force personnel were exposed to agent orange for 2 or 3 hour periods. They would go back to their base, they would shower, they would change. As far as the ground troops were concerned, they could be away from home base for weeks on end, they could be exposed to agent orange in air, food, and water; their clothing would be soaked with it; they wouldn't have an opportunity to go back and change. There is a wide range of different practice, quite apart from stress, fatigue, and the possibility of contact with other toxic chemicals, and also with tropical infections.

So to suggest that if there is any effect due to dioxin we should see it in the Ranch Hand study which constitutes a fundamental misconception of the nature and the type of exposure of ground troop personnel versus the more specialized kind of exposure in the ranch hand study.

Mr. DASCHLE. Speaking of studies, I was interested in the comment you made earlier in your testimony with regard to the wealth of information that seems to be available. As we analyze toxins and poisons in this country, we, of course, establish quite a library on each of the kinds of toxins there are.

How would you compare the information available to us on dioxin with other serious toxins that we have?

Dr. EPSTEIN. I think we have literally overwhelming data on the toxicological effects of dioxin in animal studies. To suggest that we need more animal studies is clearly an indication of unwillingness to face the issue on its scientific merits. It is an expression, to my mind, clearly of the fact that there are political and economic constraints to the interpretation of such data.

As far as the human studies are concerned, there is a remarkable consistency between the human data and the animal data. It is a remarkable consistency. The soft tissue sarcoma data is very, very similar to what we see in animals. We have seen the soft tissue sarcoma Swedish data, we have seen it in Dow workers, we have seen it in Monsanto workers, we have seen it in the NIOSH cohort study, we have seen it in Midland, Mich., and we have seen it in Vietnam veterans. What are we waiting for? What are we waiting for? We have massive data in these areas.

Now, I am not saying that if you want to spend $10 million on studying this in the next 10 years or 15 years, I am not saying you are not going to get more interesting information. But from a scientific standpoint, we have more than enough. There are overwhelming scientific precedents for a wide range of Federal regulatory actions in instances where you have far less data. Even leaving the toxicological field and just multiple sclerosis, just the cardiovascular disease in amputations, what we are seeing here is a double standard. I find it scientifically impossible to comprehend the basis for this double standard unless it masks underlying political and economic considerations for a refusal to recognize the reality of agent orange disease.

Mr. DASCHLE. Based on that wealth of data that we have—you talked about improving the legislation and you obviously find it is far too conservative for your tastes and you would like to see an additional number of diseases that would be applicable here.

Based on that information, could you touch on a few of those that you would also include in this legislation if you had the power to write it?

Dr. EPSTEIN. The fact that I am emphatically pointing out that there is a strong scientific basis of evidence for agent orange disease doesn't mean to say that I am suggesting that any veteran who comes along with any disease or any illness and who was in Vietnam should automatically be compensated. I have similar interests, as this committee obviously does, in not opening the floodgates of compensation. We clearly have to proceed in a cautious way on the one hand, but in a way that reflects equity.

Therefore, I would suggest that there should be four criteria for compensation of agent orange disease. No. 1, there has to be a history of potential exposure. I would suggest that if somebody spent 12 months in Saigon in the bars and in the agency offices and military offices in his 12 months' service there, that doesn't represent potential exposure. But leaving alone such extremes, I would say the first important point is a history of potential exposure.

The second is objective evidence of chronic disease states following exposure. Clearly, if somebody had recognizable disease before going to Vietnam, and the recognizable disease persisted after going to Vietnam, irrespective of whether he had exposure or not, he shouldn't get compensation.

Third, there has to be a consistency between the disease state for which compensation is claimed with the recognized literature on dioxin effects.

Finally, there should be an absence of other known proven causes for the disease state. If, for instance, somebody did have potential exposure and the disease occurred following the exposure, and the disease state is consistent with the recognized literature, but there are other known proven exclusive causes for the disease state, this, to my mind, would raise serious questions about compensation.

Now, within this framework, one could produce legislation which satisfied the scientific aspects of the problem, which didn't open the floodgates, and which would proceed to redress the major inequities to one of the greatest victims of the Vietnam war, namely, veterans suffering from agent orange disease.

Mr. **Daschle**. Well, I think you present a very rational approach to this, and you have given us a great deal on which to deliberate. We appreciate your testimony a great deal and I am very much grateful to you.

Dr. **Epstein**. Thank you, sir.

Mr. **Applegate**. Thank you, Mr. Daschle.

The gentleman from Mississippi, the Honorable Chairman Montgomery.

Mr. **Montgomery**. Thank you, Mr. Chairman.

I believe I will go by the rules, and I believe Mr. Edgar was here before me.

Mr. **Applegate**. Mr. Edgar is not a member of the subcommittee.

Mr. **Montgomery**. OK. Well, he'll have to wait. [Laughter.]

We'll still go by my rules, then, Mr. Chairman.

Mr. **Applegate**. Absolutely.

Mr. **Montgomery**. Thank you, Dr. Epstein, for being here this morning. You are a noted scientist and we appreciate your appearing before the subcommittee.

Let me clear up one point. You mentioned that because maybe the Veterans' Administration was not capable of making this study, that it was taken away from the Veterans' Administration and sent to the CDC in Atlanta.

Actually, Congressman Hammerschmidt and I wrote the letter to the Veterans' Administration, and we really were not critical that the VA could not do the study, but we thought it would have more credence and be more acceptable if we moved it down to CDC. So it really wasn't any criticism that the VA could not do the job and our letter didn't say that.

Dr. **Epstein**. I appreciate your tactful correction, sir.

Mr. **Montgomery**. I was reading some comments that you and Dr. Esson had made on "Nightline," where it was pointed out that well over 168 million people probably have been exposed in this country to dioxin.

My question to you is, I could understand maybe Times Beach, Mo., where you could get a level of dioxin I guess from the ground. How could you do it in Vietnam? You mentioned it was a much higher level in Vietnam.

Dr. **Epstein**. Thank you, sir. I am delighted to respond to that question.

As far as Times Beach is concerned, levels of dioxin in the soil range from 100 to 300 parts per billion. Now, it is clear that people living in those areas do have substantial potential exposure. This exposure is, however, somewhat limited by the fact that the dioxin is in the soil and there is a degree of binding of dioxin to the soil. This degree of binding is, by no means, absolute because the dioxin can be leached out of the soil by organic solvents, by other chemicals that are present there, and also dioxin is soluble in water at levels of about 200 parts per trillion and there are additional possibilities for contamination via dust and food and water. In this respect, the recent unpublished CDC study on the Times Beach residents is highly suggestive, though we don't have the full data yet, of a high incidence of abnormalities of liver function and other metabolic abnormalities.

Now, let us compare the Times Beach type of exposure with the exposure in Vietnam. You were dealing there with levels of dioxin in agent orange which were on the order of an average of 2 parts per million, but going as high as 60 or 70 parts per million dioxin. Exposure, particularly of ground troops, to dioxin-contaminated agent orange could have occurred through multiple routes, going through contaminated forests, rice paddies, and not only were there opportunities for skin contact, with herbiage and foliation still dripping with agent orange, but also contamination of air, with aerosols, of water which was highly contaminated with agent orange, or their food.

Don't forget that these men were living in the field, living in the jungles and rice paddies for weeks on end. As I stressed before when making comparisons with the ranch hand personnel, the order and the nature of the exposure was very, very different to that of the more specialized kind of exposure in the ranch hand situation.

Therefore, I would submit that while it is difficult to make precise quantitative comparisons between exposures in Times Beach and Vietnam veterans, that in both circumstances there was the potential for very substantial exposure, but exposure occurring under a wide range of different conditions.

It is interesting that there already appears to be suggestions of a consistency of some effects, between those which have been noted already in Times Beach and those which have been reported in the Vietnam veterans, with particular reference to liver enzyme abnormalities.

Mr. **Montgomery**. About what you have said, Doctor, it would seem to me the ranch hand study would certainly be important to this subcommittee in that those who handled the dioxin and agent orange had much more exposure, I would assume—and correct me if I'm wrong—than those who were in the field or near where the agent orange was being sprayed.

Dr. **Epstein**. I certainly would agree that the ranch hand study, probably designed and properly carried out, could over the course of time produce useful information. I have already mentioned that this study has been reviewed by a wide range of expert bodies and organizations, including the National Academy of Science, which I think gave a devastating critique of the study on its lack of sensitivity, quite apart from other problems and quite apart from the fact that we would need a 20- to 30-year followup before we could obtain definitive information.

Over and above that, I must stress that it is not feasible to state that because the levels of exposure in ranch hand were higher than those of ground troops, therefore, we are more likely to see effects in ranch hand than in ground troops, because the exposures were entirely different. In ranch hand personnel, the exposures were very brief, 1, 2, 3 hours. The helicopter and the plane crews would go back, they would change and be out of their contaminated clothing. On the ground, troops were exposed to contaminated materials week in and week out. Their clothing was soaked with it. Their food was impregnated with it. There was a different pattern. Therefore, to accept exposure in one doesn't mean to say you have to knock it in another. Clearly, there was exposure in ranch hand, and clearly there was exposure on ground troops, and clearly there was exposure in Times Beach. We could spend months debating the fine differences. The similarities are what is important, that there are exposures, that the exposures are common, but the patterns of exposure vary from circumstance to circumstance to circumstance.

Mr. MONTGOMERY. Thank you, Dr. Epstein. My time is up.

I would like to make one more comment. I read part of the debate that took place on "Nightline," and I am really kind of amazed that Dr. Esson, another noted scientist, and you totally disagreed on some areas of dioxin. I assume Dr. Young, who will appear before the committee in just a few minutes, will probably disagree with what you have said.

I thought maybe scientists would be closer together in reaching their decisions and their opinions on areas of chemicals.

Dr. EPSTEIN. Scientists are no better than politicians, sir, in that respect. [Laughter.]

The other point I would mention, we are dealing now with problems of toxicology and epidemiology. Dr. Esson, who is a distinguished scientist at the National Institutes of Health, was discussing this from the standpoint of basic mechanisms. With due respect, he has no recognized expertise or background or authority in the study of human toxic effects, in either animal or epidemiological clinical systems. That is the only comment I would wish to make.

Mr. MONTGOMERY. Thank you.

Mr. APPLEGATE. The gentleman from Pennsylvania, Mr. Edgar.

Mr. EDGAR. Thank you, Mr. Chairman.

Dr. Epstein, you mentioned one point in your testimony about the buildup of the chemicals. Can you talk about the exponential growth or the gradual increasing of the chemical buildup in a body? What were you trying to refer to in your statement?

Dr. EPSTEIN. What I was referring to was the fact that we have evidence from animal tests on cumulative toxicity. In other words, if you take a dose of dioxin which is enough to produce a particular effect in an animal, whatever that effect is, and if you then divide that dose into 20 or 30 fractions, and you give each of those fractions at periodic intervals of time, you will find that not only is the effect produced by the large dose mimicked by the accumulation of small doses, but the accumulation of small doses produces a greater effect than the initial large dose. This is what we refer to as the concept of cumulative toxicity. In other words, the effects produced are cumulative.

This isn't necessarily an expression of a buildup of the material in the body, but an accumulation of the adverse effects in different organ and body systems.

Mr. **Edgar**. Dr. Epstein, do you know what causes diabetes?

Dr. **Epstein**. I wouldn't regard myself as an expert on diabetes. However, as far as adult diabetes is concerned, there are a group of known risk factors, including obesity and hereditary factors.

Mr. **Edgar**. Do you think there was anything in Vietnam that might have caused diabetes?

Dr. **Epstein**. Well, I think there are data demonstrating the metabolic effects of dioxin in——

Mr. **Edgar**. How about outside of Vietnam; how about if you enter the service, you serve in the military for 3 or 4 years or whatever, is there anything in that service, apart from a war theater, that would in your opinion cause diabetes?

Dr. **Epstein**. I would find great difficulty in being able to pinpoint any factor in service which could be responsible for diabetes.

Mr. **Edgar**. Isn't it true that within 1 year after leaving the service, if you have a diagnosis of diabetes, there is a presumption that it was service connected?

Dr. **Epstein**. That is correct, sir. And this is the point I was attempting to make when discussing multiple sclerosis. The situation with multiple sclerosis lends further emphasis to your point, that if you get multiple sclerosis within 7 years after leaving the service, you get compensated.

Mr. **Edgar**. So there is a presumption for diabetes; there is a presumption for multiple sclerosis——

Dr. **Epstein**. Yes, sir, and also for this cardiovascular disease following amputation. In other words, there is a massive inconsistency in this.

Mr. **Edgar**. So wouldn't you say the presumption of those related diseases was, in a sense, a political decision, not so much based on the science, but based on the fact that there would probably be long years of extended care and, in a compassionate way, our Nation responded by presuming causality?

Dr. **Epstein**. I would say that clearly is a political decision, in the same sense as the decisions up till now not to compensate agent orange victims is also a political decision, but in the other direction.

Mr. **Edgar**. Diabetes happens to run in my family, and the genetic trait that could, in fact, affect my children and their children and so forth; then an exposure to a war situation or military service would not necessarily exacerbate that condition or, in fact, create it?

Dr. **Epstein**. I am not prepared to comment further on the specifics of diabetes. I haven't studied it. But I would agree with you, that there was a major disparity in our attitude toward diabetes, multiple sclerosis, on the one hand from agent orange disease, and on the other hand—and this disparity is all the more striking considering the massive evidence we have for dioxin and agent orange disease.

Mr. **Edgar**. If I could just ask one final question, in the testimony that is to be presented by the Veterans' Administration, in the heart of that testimony there are two sentences that use the word "suggest" when referring to studies "suggesting" a causal relationship between exposure. It is a tough word. Let me just read the two sentences that include that use of the word because I have difficulty and I want to ask the VA later whether that means there is causality or whether it is just a loose word. Unfortunately, the pages are not numbered, so let me just read.

In reference to a series of studies in Sweden using case control methods, grouped the soft tissue sarcomas together in order to investigate whether Swedish foresters and farmers exposed to herbicides and a chemical known as chlorophenol in their work suffered latent malignancies of this type. These studies have been carefully reviewed and it goes on. Then it says, ". . . and determined to have been carefully conducted and well-reported with results that suggest a relationship between herbicide exposure and soft tissue sarcomas." Then down further it says, "Investigations in the United States based on studies of industrial workers have also suggested a phenoxy compound connection with soft tissue sarcomas."

Do you suspect that in the use of the word "suggested" that the VA intends there to mean there is a connection, or that the connection is just so loose that it, you know, has little or no relationship?

Dr. **Epstein**. I am really not necessarily qualified to discuss motivation, but it seems clear that the science of the matter makes it clear that these studies demonstrate clear excesses of soft tissue sarcoma and demonstrate causality.

I would point out that we have a consistency of the soft tissue sarcomas observed in a wide range of Swedish studies, in a wide range of American studies, which again have been gone over by the National Institute of Occupational Safety and Health in their demonstration of a fortyfold excessive soft tissue sarcomas, in Vietnam veterans, and in residents of Midland, Mich.

It would appear to me that the use of the word "suggest" represents a deliberate attempt by the VA to denigrate or discredit the nature of the data which I and others find compelling, and which I believe the independent scientific community seems to find compelling.

Mr. **Edgar**. Thank you. I have no further questions, Mr. Chairman.

Mr. **Applegate**. Mr. Daschle, do you have a further question?

Mr. **Daschle**. Mr. Chairman, I think the last question that Mr. Edgar asked addressed it pretty well.

Just to reiterate, Dr. Epstein, several studies have been done, in this country and around the world, really, showing that there is a relationship between exposure and soft tissue sarcomas, at least two showing lymphomas, two with stomach cancers, and apparent high rates of testicular cancers. I am aware of a study that shows an excess of skin cancer.

Now, yesterday the Administrator said that there seemed to be a consensus of scientific opinion around chloracne and porphyrins. There seems to be quite a bit of consensus of opinion here with regard to soft tissue sarcomas.

Are there any studies that I have left out?

Dr. **Epstein**. Studies you left out in connection with what, sir?

Mr. **Daschle**. With the soft tissue sarcomas or any kind of cancer.

Dr. **Epstein**. I misunderstood. You haven't mentioned the excess of soft tissue sarcomas in residents in Midland County, Mich., close to the Dow facility. I think that has been very well demonstrated. An 800-percent excess of soft tissue sarcomas from 1970 to 1980 as opposed to the two to three decades prior to that.

Mr. **Daschle**. An 800——

Dr. **Epstein**. Yes, 800 percent. If you like, I can go into details on this.

Mr. **Daschle**. I think we are probably pressed for time, but I am grateful to you once again for elaborating on these and clarifying this part of the issue for us. Thank you again.

Dr. **Epstein**. Thank you, sir.

Mr. **Applegate**. Doctor, thank you very much for appearing before our committee.

Dr. Peter Kahn, who was to have appeared at this point in the hearings has not yet arrived. I hope he will join us later on this morning.

Our next witness will be Dr. Jerry Simmons, Veterans' Administration Medical Center, Sioux Falls, S. Dak. Welcome, Dr. Simmons.

## STATEMENT OF DR. JERRY SIMMONS, LABORATORY OF CLINICAL MEDICINE, VETERANS' ADMINISTRATION MEDICAL CENTER, SIOUX FALLS, S. DAK.

Dr. **Simmons**. Thank you.

I would like to thank the subcommittee for the opportunity to testify. I think, first of all, I am going to digress from my prepared statement in the interest of time.

One thing I should make clear, though, is that I am not an employee presently of the Veterans' Administration. I am a contract pathologist. I would like that cleared up for the record.

Mr. **Applegate**. We will note that.

Dr. **Simmons**. I have been interested in this particular field for a long time. Presently, though, just a little on my background. I am a professor of laboratory medicine, University of South Dakota, and the Clinical Chief of Laboratory Services at the Veterans' Administration Hospital in Sioux Falls, S. Dak. We have the opportunity to see many alleged agent orange victims through veterans groups and also through our pathology practice.

I trained at Ohio State Medical School and then interned at Brook General Hospital in Fort Sam Houston, Tex. During that time it was the Tet offensive in 1968 and I happened to be on orthopedic service. We saw literally hundreds and hundreds of maimed individuals that came back from Vietnam. Then in a few months I was there myself. I was a flight surgeon serving with the 101st Airborne in both Phu Bai and in Camp Eagle. I spent a lot of time in the Ashau Valley, a lot of time on patrols, perimeter patrols in our own area, and also seeing civilians in the medical-civic action program. As I mentioned, I have been on many fire bases and consider that I have a certain amount of probable agent orange exposure myself.

After Vietnam I went to the University of Michigan for my pathology residency and stayed on staff for the next several years at which time I was an employee of the Veterans' Administration hospital. As most of you are aware, in the mid-1970's we had a disaster in Michigan. There was contamination of polybrominated biphenols by a mistaken mixture of fireretardant with feed that was then given to dairy cattle. The milk subsequently went throughout the Michigan population and most Michigan residents had significant amounts of PBB in their fat.

Now, you may wonder what this has to do with my testimony. I might add that PBB and polychlorinated biphenols, PCB's, are sort of sister compounds to dioxin. I had the opportunity to work with several of the scientists at the University of Michigan on this PBB problem which resulted in several publications and symposia and presentations. I would just like to state that the problems associated with PBB that we saw were very similar to those reported both on experimented animals and in humans with dioxin, except, of course, to a much lesser degree.

I had the opportunity to do fat samples on Michigan people and we looked at levels of PBB and PCB. These were in autopsy patients. We found there were high levels of PCB's in all of the fat samples, and also quite high levels of PBB in all the fats. The distinguished gentleman from Ohio might be interested to know that we did have some Ohio samples, all of which were negative for PBB, but high in PCB.

Again, what does this have to do with agent orange? Well, it is the cumulative effects of the carcinogenic milieu that we live in. To say it can only be agent orange exposure or dioxin exposure that caused this, I think is really not scientifically tenable because all of us have so many other factors that we have been exposed to throughout our lives.

One thing I would like to suggest, something I haven't heard in the testimony so far, was that at the time agent orange was sprayed in Vietnam there was more agent orange used right here in the United States. Maybe it wasn't called agent orange but it certainly had dioxin contaminants in it. So we have a lot of people here in the United States that may be exposed to agent orange type dioxins also. Therefore, it is very difficult to find good control subjects that we can use to compare with Vietnam veterans.

I might point out that humans are lousy experimental animals because they have so many other factors you just cannot sort out. It is a bag of worms and I see no way, scientifically, that we can ever prove that our Vietnam veterans or anyone else for that matter, that the effects that we are seeing are only due to agent orange, because of all this other milieu that we have.

In that regard, I would like to remind the panel that there are many other agents that we were exposed to in Vietnam, including dapsone, which I faithfully gave to the people that were under my medical supervision and insisted they take it. And while we prevented a lot of malaria and saved a lot of lives, who knows what the long-range effects are of dapsone—it caused the deaths of 14 people due to bone marrow toxicity in combination with dioxin. For that matter, there were illicit drugs that were used of unknown composition. We don't know what the effects of those unknown compounds have been.

Also, alcohol. Most of the people that were in Vietnam drank heavily, and we don't know what the association is between alcohol and agent orange. As far as I know, no real studies have even considered looking at the combined effects of all these agents.

So when some respected scientists say there is no real evidence that agent orange causes medical problems other than chloracne, I think you have to realize that they are looking at this type of problem. We can't say agent orange alone is causing any of these symptoms because we have no one who was exposed only to agent orange.

Nevertheless, we have literally thousands if not millions of Vietnam veterans who are suffering. We can't wait for 20 years until definitive studies are done. These people need help right now.

I certainly support this bill, but I, in concert with Dr. Epstein, do not feel it goes nearly far enough. There is another problem that is perhaps even more devastating than the problems we are seeing with the physical effects of agent orange, and that is the problems of depression and the loss of libido and other things that go into the psychiatric area.

Now, I am not a psychiatrist—I'm a pathologist. But nevertheless, the people I have come in contract with all express very similar stories which at least seem to be some type of delayed effect from Vietnam.

Another thing to consider is that in the Veterans' Administration we see roughly 10 percent, if that many, of the veterans. There is 90 percent out there that we don't see, and a lot of those people were exposed to agent orange. I have talked with many of them. When you really get to talking with them—and I am talking now about successful people, bank vice presidents, financial advisers, the cream of our society—and those that have been to Vietnam, when you really get to talking with them, you realize there is something radically changed in their lives and all of them are suffering to some extent or another of what is now known as post-Vietnam stress—post traumatic stress syndrome. I think this is another issue that has to be addressed at some time by this committee.

Now, if I may, I would like to comment on some of the studies that are going on. I have been in contact with Dr. Irey of the AFIP. As you know, the Veterans' Administration is, in conjuction with the AFIP, studying pathologic tissues from agent orange-exposed veterans. The first study, which has not yet been published—I believe there are copies available to the committee—does not show any real clustering yet of types of cases of any kind from Vietnam. I do not have the exact data on that.

But I would like to say that the Veterans' Administration—and we're all at fault on that—is not really gathering the data. There is a lot of data out there that does not go into those files. I also would like to state that there are probably hundreds of other cases, and I know of several, that have never been to the Veterans' Administration, various types of cancers, various types of strange immunologic, strange cardiovascular diseases that are unheard of in young men.

Most of these reports are anecdotal, they are not well controlled, so scientifically they do not stand up. However, if we could get a good, honest, cooperative study such as only the Veterans' Administration is capable of doing, I think we might be able to get some answers.

Again I would like to stress that we don't have the time to wait for this type of data. Let me remind you of the asbestos exposure of the World War II shipyard builders. The problems from that exposure are only now becoming known. The same with radiation exposure after the atomic bomb testing. It is 20 or 30 years later, and only now do we know that there is a real problem with this, the increased incidence of leukemia, with asbestos there are increased problems with lung disease.

In summary, I think the answer to this has got to be a political and socioeconomic answer. If I may indulge, I would just like to have 1 more second here. I have been with the Veterans' Administration now for almost 14 years. If you count the military experience, it is well past 15. I would like to say they are underfunded. They are asking all of us, and you are asking all of us, to do more and more with less and less. All of our physicians are overworked. When we see our agent orange people coming in, it is very difficult to show them the compassion, give them the time that they need. Most of the physicians I talk to say "If I only had the time I could spend with these people, to get to know them and take their histories properly." But we are so rushed with all the tremendous workload that we have that there is no way you can adequately examine these patients and adequately take care of them. I think this has to be addressed.

The Vietnam veteran, including myself, feels betrayed. We feel betrayed by our country, we feel betrayed by the Veterans' Administration, and we feel that there should be something done. We, indeed, need to be welcomed home and we need to reconcile the problems that we have had.

I am open for questions.

[The statement of Dr. Jerry Simmons appears on p. 354.]

Mr. **APPLEGATE**. Thank you, Dr. Simmons, for your statement and your lobbying efforts. We appreciate both very much.

I just had a couple of questions. You had stated that there were a number of dioxins sprayed in the United States. On page 5 you say most Americans seem unaware that during the period 1961 through 1969 44 million pounds of 2,4,5-T were sprayed in Vietnam, while 78 million pounds were used in the United States, and that it should be pointed out military spraying utilized 27 times more herbicide per unit area than most of the domestic operations, which is very concentrated.

Given that concentration, and with reference to your comments on the various studies, I would like your comments on the "Ranch Hand study," because it seems to be revolving around a question of the amount of concentration of dioxin and exposure and whether or not those who were exposed to agent orange presently being diagnosed and studied in the "Ranch Hand study" offered from any varied degree of effects based solely upon dioxin concentration and amount of exposure.

Dr. SIMMONS. I was there; I was in the field; I know the conditions that existed in the field and also that existed in the Air Force units, at least the ones that I visited. Believe me, it was heaven to be in the Air Force, which I wasn't.

I think the Operation Ranch Hand will give us some valuable data in spite of the previous criticisms, but I think you have to take it for exactly what it is. It is a study of Air Force personnel that handled the agent orange, and hopefully they were somewhat instructed to be careful with how they were handling it. This is in complete contrast with the people that were out in the field and at the fire bases and various other places that drank the contaminated water, that literally lived and wallowed in this contaminated area for weeks, if not months, and whether you're in a base camp or wherever, the sanitary conditions were deplorable.

Again, I stress that the people in the fields in particular were exposed to all kinds of other things. In my unit everybody took Dapsone. Several units were sprayed with agent blue, which is not addressed. There are other kinds of code-named agents that were used in Vietnam and need to be covered by this type of legislation.

Mr. APPLEGATE. Do I understand, Doctor, that when you're saying the bill doesn't go far enough, you're talking about the fact that it isn't as all-inclusive as it ought to be with regard to the contaminants themselves, or just in relationship to the herbicide agent orange or that it doesn't go far enough in the diseases that have been caused by it. Are you also saying that we presently have sufficient scientific evidence upon which to base and consider in finality this legislation?

Dr. SIMMONS. To answer your question, yes, I don't think it goes far enough in either way. I think it is really deplorable that this bill is necessary at all. In a way, I guess I agree with the DAV, that there is no way this bill should even be here. If it had been handled appropriately in the first place, using scientific evidence, there would be no reason for this bill. Those people who had problems would be duly compensated, duly taken care of. Their health care needs would be met and we wouldn't be here today.

But, unfortunately, that has not occurred, at least in the minds of the Vietnam veterans and in the minds of Congressmen and people in the United States, so therefore we have this bill and I certainly support it.

Mr. APPLEGATE. So what you're saying is there should be an accepted presumption that these diseases have been caused, as others have been caused, by the activity and the duty in the service?

Dr. SIMMONS. Right. I know of several cases, again anecdotal, but I know of several cases of people who have died from kidney cancers. I have a young man right now we're taking care of in his early thirties who has multiple myloma. That's a disease of old people. It is almost unheard of to have it that young. It has been reported, but it's very rare.

Under this bill, he would not be eligible for compensation, and yet because of the disease he has had back problems and he cannot work. He wants to work. He doesn't want to be compensated for agent orange. He would rather go out and work and be a productive member of society, but he cannot.

Mr. APPLEGATE. Thank you very much, Dr. Simmons.

The gentleman from South Dakota, Mr. Daschle.

Mr. DASCHLE. Thank you, Mr. Chairman.

Dr. Simmons, thank you for your testimony.

One comment that you made at the beginning that I think may need clarification. The impression I had from Dr. Epstein's testimony was that he was pointing out there is a much higher incidence of some of these diseases, of course, with higher exposure levels, given the fact that there are exposure levels that perhaps all of us are susceptible to, but that the exposure level is higher with resulting higher incidences of medical problems.

Let me ask you about analogous situations within the VA today. Obviously, we have quite a rich history of providing presumable compensation for other diseases. Let me ask you what medical consensus there seems to be on cardiovascular disorders. Is there a body or a wealth of information that, once a person suffers from amputation, there is a causal relationship between amputation and cardiovascular disease?

Dr. SIMMONS. That's a very difficult question to answer. I don't think the good studies have even been done on that yet.

Mr. DASCHLE. No studies have been done, except for one, as I understand it. I understand, of course, the DAV has also done some kind of a report with regard to this whole issue.

But what you are saying is, there is no consensus within the medical community that having an amputation brings about cardiovascular problems?

Dr. SIMMONS. Not per se.

I might just add one thing that is not too well appreciated because we're all thinking chloracne, we're all thinking porphyria, we're all thinking soft tissue sarcoma—and Dr. Epstein brought this out—and that is that there is definitely an elevated level of lipids. We're not even testing for that in our agent orange exams most of the time. There is an elevated level of lipids, as demonstrated by the Czechoslovakia study and several others, where there is definitely elevated chloresterols. Again, no scientific studies have been done to my knowledge. But we are getting reports on an individual basis of very strange vascular diseases such as strokes occurring in young men—again, an old man's disease.

I saw a young man in Ann Arbor when I was there and something happened to his vascular system. His hands died, and his feet died. They had to be amputated. Eventually his whole body died. And when I did his autopsy and attempted to send some fats in, because I knew he was an agent orange exposed Vietnam veteran, there was no place I could get any dioxin levels done.

That is another matter. At the present time we do not have the facilities, or maybe just now have the facilities, to look at the dioxin levels in human fat. The collection of fat in the past has been discouraged.

As I was bringing out with Dr. Irey's study, there is still a wealth of data that is not being used because we have cases since the 1970's and even before in the VA of various types of tumors and other conditions these Vietnam veterans have had that are not in the agent orange registery in the tissue submitted to AFIP.

When you talk with other people in other places—I have the opportunity to meet with the VA chiefs at various meetings of pathology, and they are not sending in all their agent orange cases. Until just recently, there was no way we could even identify those patients. Now, at last, they have given us an identifier on the veterans' cards. So it is very difficult.

If I may digress once more, the statement was made that if there was no evidence Vietnam veterans had problems in the service, and if there wasn't anything on their medical records, that they wouldn't be compensatable. This bill goes beyond that, of course. I would like to point out that many records were lost in the fire—including my own, by the way—at Levenworth many years ago. There is no way we can go back and check these people.

Mr. DASCHLE. Let me ask you the same question with regard to diabetes and multiple sclerosis. Is there a wealth of information, have studies been done to show the relationship between that disease and service in which that disease may have been contacted? Is there a consensus of opinion within the scientific community that that is a presumptive disability based upon any causal relationship?

Dr. SIMMONS. No, not that I'm aware of. In fact, as Dr. Epstein pointed out, if you are talking about scientific evidence, there is no doubt that the evidence is overwhelming that not only the diseases that are addressed in this bill but other diseases such as depression and psychiatric disturbances have been associated with agent orange or dioxin exposure in industrial areas——

Mr. DASCHLE. So from your point of view—we're out of time—but from your point of view, the wealth of information already available on exposure to agent orange and the relationships between exposure and certain diseases is substantially greater than the information currently available between whatever service connection there might be to the medical diseases in diabetes, MS and cardiovascular disease; is that what you're telling me?

Dr. SIMMONS. Certainly.

Mr. DASCHLE. Thank you very much, Dr. Simmons.

Mr. APPLEGATE. Thank you, Mr. Daschle.

The gentleman from Mississippi, Mr. Montgomery.

Mr. MONTGOMERY. Thank you, Mr. Chairman.

Thank you, Dr. Simmons, for being here today. I was interested in your statement of the exposure to agent orange by so many Americans, especially in the farming areas. I assume that is correct, where they do use certain types of chemicals that have dioxin?

Dr. SIMMONS. Yes, and railroad rights-of-way and other places.

Mr. MONTGOMERY. I was also interested in your statement about many other problems besides agent orange. I made this statement yesterday, and it was pointed out in the Australian study on agent orange, that after the study was made it checked out that the Australian veteran was showing up with more problems other than agent orange, such as you mentioned, psychological problems that developed and for some reason quick tempers.

In the report they said you ought to concentrate more on the psychological areas that really seem to be developing more than the agent orange tests. Is that basically what you were saying?

Dr. SIMMONS. One thing I didn't bring out, if I just may digress, because it has something to do with this scientifically. When we measured PBB levels in experimental animals at the University of Michigan, we found PBB very early in the brains. We could not find it later on a chronic basis, but the early tests showed PBB in the brains. Now, I am not aware of any tests that shows this with dioxin, but certainly it is a sister compound. I don't know if it has even been looked at in that particular aspect.

The literature is voluminous and nobody can cover it all. However, there is also evidence that a similar type of problem as far as quick tempers, depression, this type of personality change has occurred following these industrial accidents. I say that there is really no evidence that this is due to something other than agent orange. I know of no real good controlled studies where people have even looked at this yet.

I would like to say there is no doubt—I think no one will dispute the fact that Vietnam veterans are hurting. Their problems are real. I think we're just having a smoke screen on whether they are due to agent orange or not. I think that is completely irrelevant. We have to take care of these people that have served their country. I think that's what this is all about.

Mr. MONTGOMERY. I certainly don't have any problems with that statement. What we need to do is find out where the major problem is with the Vietnam veteran and gear our resources to that problem.

I don't guess you're saying psychological or quick temper problems are showing up in the Vietnam veteran who had more exposure to agent orange. There has been no study of that. What I am reading is that just the Vietnam conflict, the problems back here at home, have caused a lot of these psychological situations.

Dr. SIMMONS. Except, sir, that most of these people, at least that I have been aware of, do admit to agent orange exposure. I would like to point out that the literature clearly shows that this same type of behavior is present in dioxin-exposed people in the Italian incident and several others. So presumptive evidence being the same with sarcomas and other things, I think we also have to consider this aspect of Vietnam veteran problems.

Mr. MONTGOMERY. Thank you.

Thank you, Mr. Chairman.

Mr. APPLEGATE. Thank you, Mr. Montgomery.

The gentleman from Pennsylvania, Mr. Edgar.

Mr. EDGAR. Thank you, Mr. Chairman.

Earlier we talked about the fact that other diseases were, in fact, given the presumption of causality. Do you have the same feeling that Dr. Epstein had, that we ought to respond very quickly on this?

Dr. SIMMONS. Absolutely. I echo his feelings. What are we waiting for?

In almost any other field, if the FDA had this kind of evidence on a drug, it would be off the market in seconds. What are we waiting for? I just don't understand it, from a scientific standpoint, from a human standpoint. What are we waiting for?

Mr. EDGAR. Thank you. I have no further questions.

Mr. APPLEGATE. Thank you very much, Dr. Simmons, for appearing before the committee.

Now, if I may ask again, is Dr. Peter Kahn in the hearing room? [No response.] Yes, Dr. Simmons.

Dr. SIMMONS. If I may, I believe he had a meeting with Mr. Walters and will be in shortly.

Mr. APPLEGATE. Next we have the Veterans' Administration, represented by Miss Dorothy Starbuck, Chief Benefits Director; Dr. Donald Custis, Chief Medical Director; Dr. Al Young, specialist in environmental science; and Mr. John Murphy, Chief Counsel.

Miss Starbuck and gentlemen, we appreciate, as always, your presence and the expertise which you share with the various subcommittees of the Veterans' Affairs Committee. We are looking forward to hearing what you have to say. You may proceed at any point.

## STATEMENT OF DOROTHY STARBUCK, CHIEF BENEFITS DIRECTOR, VETERANS' ADMINISTRATION, ACCOMPANIED BY DR. DONALD CUSTIS, CHIEF MEDICAL DIRECTOR; DR. ALVIN YOUNG, SPECIALIST IN ENVIRONMENTAL SCIENCE; JOHN MURPHY, GENERAL COUNSEL; AND DR. BARCLAY M. SHEPARD, ACTING DIRECTOR, AGENT ORANGE PROJECT OFFICE

Miss STARBUCK. Thank you very much, Mr. Chairman. I am very pleased to be here this morning to present the views of the Veterans' Administration on H.R. 1961, of the 98th Congress.

Mr. Chairman, we in the VA share with you and other Members of Congress the desire for a meaningful Federal response to the fears of veterans who served in Vietnam, that their exposure to agent orange may have long-term adverse effects on their health. However, we consider the approach taken in H.R. 1961 inadvisable given the present state of scientific knowledge.

The controversies arising from the Government's use of agent orange in Vietnam are not yet resolved. Before turning to the Veterans' Administration's observations concerning the several issues raised by H.R. 1961, I would like to emphasize that the potential cost of paying compensation based on any agent orange-caused disabilities played no part in our deliberations on this measure. The Federal Government, since its beginning, has fulfilled its obligation to veterans disabled in the line of duty and will continue to do so.

Mr. Chairman, the devastating wars of this century, and the need to maintain peacetime forces in order to assure the defense of our Nation, have been accompanied by legislative and programmatic developments intended to assure that no veteran's reasonable claim to compensation is denied. This is true whether the disability results from a combat wound, service-incurred disease, in-service accident, psychological trauma resulting from combat or other conditions of military service, or exposure to a substance known or later discovered to have adverse health effects.

We are proud of our agency's record of achievement. It can safely be maintained that our compensation program is the finest in the world, both in terms of the number of veterans we serve and in the amount of benefits paid. Moreover, the American people who fund this program with their taxes have given it overwhelming support, as has the Congress of the United States.

The preservation and integrity of the compensation program are among the highest priorities of the Veterans' Administration.

There are certainly many veterans suffering from illnesses they ascribe to exposure to that herbicide, especially its contaminant dioxin. Although scientific evidence is lacking, there are persons in the medical and scientific communities who contend that exposure may lead to a host of disorders that appear long after the exposure has ceased. There are also organizations and individuals who believe very sincerely that the Veterans' Administration has not responded adequately to the issues involved.

As guardians of the public trust, Congress and the administration share, I believe, a commonality of aims respecting these issues. The compensation program must be attuned to justifiable conclusions about the connection between agent orange exposure and disorders possibly arising from that exposure. At the same time, we must do our best to avoid taking steps that have the potential for undermining the program's credibility and legitimacy because of inconclusive scientific evidence. I know that you and other members will give careful and thorough consideration to the bill, keeping in mind the commonality of aims to which I have alluded.

H.R. 1961 is intended to assist veterans who served in Southeast Asia during the Vietnam era establish entitlement to service-connected disability compensation if they are currently suffering from one of the disorders specified in the bill. It would do this by amending section 312 of title 38, United States Code, in order to provide for a special presumption of service connection applicable only to these veterans.

Mr. Chairman, the bill is based on the premise that each of the specified disorders, no matter how long after military service symptoms appear, can be attributed to exposure to a phenoxy herbicide in service. During the period 1962 to 1971, phenoxy herbicides, including agent orange, were used in Vietnam. As I have noted, H.R. 1961 is an effort to respond to the widespread concern that exposure to agent orange, especially its contaminant dioxin, may have long-term adverse effects on veterans' health.

Authority to award compensation on the basis of the presumption provided for in the bill would terminate 1 year after submission to Congress of the comprehensive epidemiological study mandated by Public Law 96-151. This sunset provision is analogous to the sunset provision applicable to VA health care for certain disorders possibly associated with phenoxy herbicide exposure, authorized by Public Law 97-72. Both sunset provisions recognize the current uncertainties as to the long-term effects of exposure.

The agent orange controversy, as it relates to individual veterans' compensation claims, involves two basic questions: One, whether the veteran was exposed, and two, whether the veteran's disability results from that exposure. H.R. 1961, it should be noted, does not require any evidence of exposure; it would afford the presumption to any veteran who served in Southeast Asia during the Vietnam era, 1964 to 1975.

We have previously made public our decision to resolve the issue of exposure in a manner favorable to veterans. Unless there is affirmative evidence to the contrary, we are prepared to presume exposure if a veteran served in Vietnam during the relevant period. This policy, prompted by the lack of a definitive method for identifying exposed individuals, is consistent with our long-standing policy of giving veterans the benefit of the doubt.

There may be, however, some cases in which affirmative evidence refutes even the possibility of exposure and, therefore, our policy is necessarily qualified. The lack of any similar qualification in H.R. 1961, in our view, is unjustifiable. We observe also that affording the presumption to veterans who served in Southeast Asia—a far broader region than Vietnam, embracing areas where no phenoxy herbicides were used—inappropriately expands the category of veterans intended to be benefited.

Our principal concerns, however, relate to the concept of an open-ended presumption that would be established by the bill and to the conclusions it embodies as to the specific disorders chloracne, porphyria cutanea tarda, and the several malignancies grouped as soft tissue sarcoma.

The bill would also authorize presumptive service connection for additional disorders, provided for by regulation, that medical research has shown may be attributable to chemical exposure or environmental hazards or conditions. This requirement is vague—rules creating such presumptions should be based only on well-accepted, scientifically valid findings—and also unnecessary in view of the Administrator's current authority to issue regulations.

The post-service presumption periods provided for in section 312(a) of title 38 are appropriate for chronic diseases whose inception in service may not be recorded because the development of pathology is gradual and insidious. They are justifiable, Mr. Chairman, when reasonably supported by medical knowledge as to the pathological courses of the particular diseases.

Congress has wisely set time limits on these presumptive provisions. Unless symptoms of the disease appear within a specified period of time after service, the presumption is not available. The section 312(a) presumption, together with the time limits, assures that no veteran's reasonable claim is overlooked, but also does not dictate grants of service connection when there is no evidence of service incurrence and it is not reasonable to infer service origin.

Reputable studies have concluded that dioxin exposure may result, within a relatively short period, in chloracne. PCT resulting from exposure also appears within a few weeks. On the other hand, no studies have shown that exposure results in the initial appearance of these disorders after lengthy delays.

Mr. Chairman, our current authorities are adequate, without the need of a presumption, to award service connection and compensation, if appropriate, in cases of chloracne or PCT appearing within expected time limits after the exposure. Requiring us to award service connection for initial occurrence of these disorders long after the exposure incidents is, we believe, unjustifiable in the absence of any evidence indicating they are latent effects of exposure.

As I have noted, individuals in whom these relatively rare disorders appear begin to suffer symptoms soon after exposure, ordinarily within days or weeks. Chloracne is a skin disorder caused by exposure to certain chlorine-containing chemicals, including dioxin. In its more serious manifestations, it causes discomfort and disfigurement. Most cases clear up within a year or two after the exposure ceases, but in a few, the disorder persists. The Veterans' Administration acknowledges that chloracne can result from exposure to agent orange during service in Vietnam and has established procedures to assure careful and liberal consideration of all claims based on this disorder.

Since 1978 we have awarded service connection in 1,225 skin disorder cases involving veterans who served in Vietnam. We have scrutinized more than 3,000 claims for service-connected benefits to determine whether there are indications of chloracne. Those cases in which it was believed this diagnosis was at least possible were further reviewed by a VA dermatologist, and 13 have been examined in person by dermatologists at prestigious private clinics.

Although all of these cases involve skin disorders of various types, and all involve veterans who served in Vietnam, only one case of possible chloracne has been identified. We will, of course, continue our investigations of this issue.

H.R. 1961 would also extend presumption of service connection for chloracneform lesions. This is a term not found in medical or scientific literature but can be taken to mean "lesions resembling chloracne." As certain common skin disorders may resemble chloracne, this term is overly broad and would, we believe, cause unnecessary confusion.

PCT, an uncommon liver disorder, can be triggered by exposure to various chemicals, including alcohol. There is no evidence that PCT is a latent effect of exposure. Each attack ordinarily subsides in about a year after contact with the chemical ceases, but prolonged exposure, as in chronic alcoholism, may cause permanent damage to the liver.

An attack of PCT induced by agent orange, or exposure to any other chemical during service in Vietnam years ago, would not be expected to impair a veteran's health today. As is the case with chloracne, we regard our current authorities as fully adequate to assure proper consideration of PCT claims based on exposures during military service. As a technical matter, the proper application of section 313 of title 38, United States Code, making section 312 presumptions rebuttable if there is evidence of an intercurrent cause, would reduce the likelihood of awards of service connection based on the PCT presumption, if enacted.

Mr. Chairman, the issue as to whether the malignancies grouped as soft tissue sarcomas result from phenoxy herbicide exposure presents a problem of far greater complexity. There is considerable uncertainty in the scientific community on this issue. Advocates of the belief that exposure causes soft tissue sarcoma generally cite studies involving cancer victims believed to have been exposed to phenoxy herbicides whose first symptoms appeared long after the exposure. Because it is well established that exposure to radiation and other agents like asbestos and benzene may result in the latent development of malignancy, these advocates reason by analogy that phenoxy herbicide exposure causes soft tissue sarcoma.

The vital question is, therefore, the weight that should be given to the studies they cite.

Soft tissue sarcomas are a group of malignant tumors, or cancers. Any sarcoma arises in a body cell that does not cover a body surface, form glandular tissue, or line certain body cavities. Soft tissue excludes sarcomas in hard tissues such as bone or cartilage. Hence, soft tissue sarcomas arise from such body tissues as muscles, tendons, blood vessels, fat, and connective tissues.

Certain cancers share some characteristics of soft tissue sarcomas but are not placed in that group. These include most brain tumors and the so-called blood cancers, chiefly the leukemias. Some authorities include tumors of the lymph nodes, the lymphomas, with the soft tissue sarcomas. The World Health Organization "International Classification of Tumors, No. 3, Histological Typing of Soft Tissue Tumors," however, excludes lymphomas and appears to be adequate for purposes of defining the malignancies in this category.

There is no evidence that all soft tissue sarcomas have a common etiology or cause. These malignancies differ from one another as to how rapidly they grow and spread, how they are treated, and the results that treatment achieves.

These malignancies are rare. According to the National Cancer Institute, they comprise 2.76 percent of all cancer cases in men aged 25 to 29, and 0.58 percent of all cancer cases in men aged 55 to 59. The percentage declines because other types of cancers become increasingly common with age. Lymphomas, sometimes included with soft tissue sarcomas, contribute another 5.21 percent at ages 25 to 29, and 2.40 percent at ages 55 to 59.

Although there is no evidence establishing a common cause for these sarcomas, some malignancies in the group are known to be associated with exposure to environmental hazards. For example, malignant mesothelioma is known to be caused by asbestos exposure, and angiosarcoma of the liver by exposure to vinyl chloride.

Because these malignancies are rare, it is difficult to devise adequate techniques to investigate their causes. A series of studies in Sweden using the case/control method grouped the soft tissue sarcomas together in order to investigate whether Swedish foresters and farmers exposed to herbicides and a chemical known as chlorophenol in their work, suffered latent malignancies of this type.

These studies have been very carefully reviewed by Richard Remington, dean of the School of Public Health, University of Michigan, at the request of the Office of Technology Assessment and determined to have been carefully conducted and well reported with results that suggest a relationship between herbicide exposure and soft tissue sarcomas. Significantly, Dr. Remington pointed out the limitations of the case/control methodology and found the Swedish studies inadequate to permit definite conclusions.

Investigations in the United States based on studies of industrial workers have also suggested a phenoxy-compound connection with soft tissue sarcomas. In addition, an East German investigation of malignant neoplasms among pesticide sprayers and agricultural technicians tends to support the Swedish studies by finding a single case of soft tissue malignancy which probably was a soft tissue sarcoma.

Other studies in Finland, New Zealand, Great Britain, the Netherlands, and Italy have not confirmed the Swedish studies. In addition, a separate investigation of Swedish forestry workers casts some doubt on the Swedish studies.

Mr. Chairman, we do not disagree with Dr. Remington's conclusions as to the credibility and limitations of the Swedish studies. They lay a predicate for further investigation and do not rule out the possibility of a causal link. They do not, however, provide a reasonable basis upon which to favorably decide VA compensation claims.

We recognize the importance of careful scientific analysis in matters of this kind, and I have attached to my prepared statement detailed background papers concerning these diseases.

The comprehensive epidemiological study mandated by Public Law 96-151, together with other ongoing studies including some devoted specifically to the soft tissue sarcoma issue, may resolve many of the controversial questions raised by the use of agent orange in Vietnam. As I stated at the outset, we must work toward the dual objective of fair compensation for any agent orange caused disabilities and avoidance of steps that would compromise the integrity of the program. At this point, there is no evidence that either chloracne or PCT is a delayed effect of exposure, and we believe the provisions of H.R. 1961 respecting these disorders are not justified. We do not believe it has been satisfactorily demonstrated that exposure can cause soft tissue sarcoma. Accordingly, we oppose the enactment of H.R. 1961.

In view of the current state of scientific findings, enactment would compromise the integrity of the compensation program and engender unfounded fears among Vietnam veterans that lethal illnesses may yet befall them as a result of having answered duty's call. Our biding moral obligation to veterans who have given so much demands that we act responsibly in all matters affecting the compensation program.

Mr. Chairman, that concludes my prepared remarks. My colleagues and I will be pleased to answer any questions.

[The statement of Dorothy Starbuck appears at p. 360.]

Mr. **Applegate**. Thank you very much, Miss Starbuck. As always, you have a very well-prepared statement and we certainly appreciate your being here.

I just have one short question, and then I'm going to let the others have an opportunity to ask questions. It has to do with a comment appearing on page 5 of your testimony. You state that the bill we have before us, H.R. 1961, "does not require any evidence of exposure." Then, of course, throughout your testimony you disclaim all available information with regard to causes of the diseases involved in the bill, questioning the studies while trying to make your point as valid as you possibly can.

You say there is no evidence of exposure and, therefore, "it would afford the presumption to any veteran who served in Southeast Asia," yet, at the same time, isn't that what we're doing with all of these other diseases? For example, a veteran comes back with cardiovascular disease or with some other type of diseases, and while there really isn't any type of service connection upon which to establish causality, we do this just the same.

Miss **Starbuck**. With respect to the chloracne or the skin disorders, some of the skin disorders, and the PCT, we have conceded exposure in Vietnam only, not to the Southeast Asia area. What you say is true, that someone serving in the Southeast area may contract a disease or incur a disability in that area without ever having been exposed to agent orange and may claim compensation for the residuals of that disease or injury and there would be no question of exposure with respect to the decisions that were made in the agency on that claim.

Mr. **Applegate**. I appreciate that, but this strikes me as operating under a double standard.

Mr. **Edgar**. Would the gentleman yield?

Mr. **Applegate**. Yes.

Mr. **Edgar**. I wonder if you could answer the question that was raised earlier, which is at the gentleman's specific point. What is it about diabetes that gives us the opportunity to determine causality? What is it about multiple sclerosis that gives us the presumption of causality as a war-related or service-connected disability to permit the ability to expend public funds for those things? Is there better data in relationship to service connection with diabetes and multiple sclerosis than we have had in the testimony and in the data on agent orange?

Miss **Starbuck**. Mr. Edgar, the relationship as it has been set out in section 312 for specific diseases, provides that if a disability manifests itself within a year to a degree of 10 percent or more, we will presume that that disability arose during a period of military service.

Mr. **Edgar**. What about the 7 years on multiple sclerosis? Is that wrong?

Miss **Starbuck**. That is not wrong, sir. The change from I believe from what had previously been 3 years to 7 years was on the basis of medical considerations that this is a disease that is a long time manifesting itself from the incident which triggers that disease. I would ask perhaps if Dr. Custis could add something to that response.

Dr. **Custis**. It strikes me that the problem here is a basic understanding that any disability, whether it be diabetes, cardiovascular problems, a traumatic incident in an automobile crash, during active duty is compensatory, without having to address whether or not the duties assigned were directly of an etiologic nature in the development of a specific disability.

So we are not talking about presumption in that case. Any disability that occurs during active duty is compensable. Now you get into how long do you carry that concept into postactive duty years, and that's where the confusion starts coming into the picture.

Could I ask the forebearance of the committee just to take a couple of minutes to make sort of an opening observation of my own?

Mr. **Applegate**. Certainly, Dr. Custis. Go right ahead.

Dr. **Custis**. Sitting here this morning, I can't help but be reminded of the story of the blind man, each of us having a hold of a different extremity, the appendages of the elephant. Why so much disagreement as to what we've got a hold of? Well, for one thing, we are all a little blind. There is an awful lot of the anatomy that we still have to find out about. And then it is very tempting to make a selective use of information. I think we have seen this morning a masterful display of that technique, and it was delightfully entertaining.

We respect the right of those who disagree with us to have their opinion, and we do not question their sincerity and motivation and we would hope that they, in turn, would find it within themselves not to disparage ours. We, along with other Federal agencies, now, for several years, have felt it our responsibility to track the literature. We have done so in depth and, in doing so, we have tried hard, conscientiously, to maintain a dispassionate, objective, unbiased perspective. It hasn't been easy because we all share compassion and empathy for the sick veterans.

But considering all the literature, we feel that the suggestive evidence from some case/control studies simply lacks corroboration. There is as yet no consensus within the scientific community that a causal relationship has been established between dioxin exposure and delayed health problems experienced by some Vietnam veterans. What is more, this opinion is shared, among others, by the Council on Scientific Affairs of the American Medical Asociation and by the British Ministry of Public Health.

When will it come? When will we get consensus? We don't know. But it will take a set of circumstances as follows: Consensus will hopefully be achieved when epidemiologic data is statistically significant, when research findings proving a causal relationship can withstand the scrutiny of peer review, and when research results can be duplicated by other investigators.

Just a couple more minutes to quote from those two authorities to which I alluded. The AMA Advisory Panel:

> While 2,4,5-T and 2,4-D herbicides have been used in agriculture, forest management, and residential landscaping for over 30 years, there is still no conclusive evidence that they or TCDD are mutagenic, carcinogenic, or teratogenic in man, nor that they have caused reproductive difficulties in the human.

On February 7, 1983, the British Minister of Agriculture, Fisheries, and Food presented to the House of Commons the final report of the Advisory Committee on Pesticides, on the safety of phenoxy acid herbicides. Quoting from that report, he said:

> In the light of available medical and scientific evidence, we conclude that present formulations of 2,4,5-T herbicides or, for that matter, other phenoxy acid herbicides, do not pose a safety hazard whether used in agriculture, forestry, or elsewhere.

Finally, the British report referring to the soft tissue case/control studies from Sweden. The committee report considered their epidemiologic procedures as being "unorthodox" and their data "unreliable." Their report noted, furthermore, that a similar case/control study from New Zealand and cohort studies of herbicide users in Sweden itself and in Finland identified no cases of soft tissue sarcomas.

That for openers, Mr. Chairman.

Mr. APPLEGATE. I would like to see your closing, sir. [Laughter.] Thank you, Dr. Custis.

Let me just ask you one thing. I still find it difficult to understand the requirement for evidence while other disabilities and diseases do not appear to require that same measure of causality.

Have you estimated what the first-year cost would be if this bill was passed?

Miss **Starbuck**. Mr. Murphy, do you have those costs?

Mr. **Murphy**. Mr. Chairman, the estimated cost I would have to give as a range because of the uncertainty as to what is included within the definition of soft tissue sarcoma, principally along the lines of whether lymphomas are included or not.

The range in compensation costs, the estimate would be $2 million to $11 million, and administrative costs would be $6.2 million. That is a fiscal year 1984 estimate.

Mr. **Applegate**. $2 to $11 million, that range, and——

Mr. **Murphy**. Excuse me. In compensation, it would be $2 million to $11 million. In DIC, it would be an additional $3.5 million to $18.7 million. The administrative costs would remain at $6.2 million.

Mr. **Applegate**. This is for fiscal year 1984, correct?

Mr. **Murphy**. It would be a range of approximately $5.5 million to approximately $30 million. I just totaled those up in my head, Mr. Chairman.

Mr. **Applegate**. Thank you very much. I appreciate that.

I am going to turn the questioning over to my colleague from South Dakota, Mr. Daschle.

Mr. **Daschle**. Thank you, Mr. Chairman.

Dr. Custis and Miss Starbuck, I don't even know where to begin. You talk about skewed information and singling out a certain set of statistics. I think you have proven that there are those within your administration that are equally, if not more, able to manipulate those facts and figures than are those witnesses who came before us earlier.

But before we even begin to talk about causal relationships, let me ask you a question with regard to the law. Does not law now require simply the manifestation of the disability, not the cause?

Miss **Starbuck**. That is correct, sir.

Mr. **Daschle**. I am glad to straighten that out initially.

Mr. **Murphy**. During the period of service.

Mr. **Daschle**. That's right.

Let me ask you, can you tell me about the causal relationship that will exist in a veteran when he comes for presumptive disability on MS?

Mr. **Murphy**. I can tell you what it is based on in law, it is because of what is medically known of the nature of the illness and the period that it is logical and reasonable that its incidence would have occurred——

Mr. **Daschle**. Logical and reasonable. But there is no data, no base of information that everyone can turn to and say "this is the reason why we can establish that causal relationship." There may not even be a consensus of opinion, is that correct, on what causes MS?

Do you know what causes MS?

Mr. **Murphy**. We are talking about the relationship, the nature of the way the illness develops and manifests to the period of military service.

Mr. **Daschle**. That's the point. I think you can say this in every one of the cases, that you know as well as I do that there are no causal relationships between MS and the service. There are no causal relationships between diabetes. There are no causal relationships in cardiovascular disease. And yet we have already assumed, or presumed, disability in these cases and have provided compensation.

Now, let me ask you this——

Mr. MURPHY. Mr. Daschle——

Mr. DASCHLE. Let me finish, if you will.

I am interested, too, in this whole question of the reservoir of information that currently exists. As we look to those three diseases, every witness that has come before this committee so far this morning has said the preponderance of information currently available which establishes a causal relationship between these three diseases and exposure to dioxin is insurmountable in comparison to these other three diseases.

Now, we talk about some of the information made available, a five to sixfold greater risk in developing soft tissue cancers, there exists occupational data in this country showing a fortyfold increased risk in exposed workers, and an 800-percent increase in soft tissue cancer risk. We have five different sources on soft tissue sarcomas apparently from testimony this morning, and yet you're saying you can't service connect.

Now, on the basis of one study that I am aware of, you have made a similar presumption for cardiovascular disease. Granted, they have to be service connected. But I can't see the causal relationship. Even though it is not necessary, you are basing your whole argument on causal relationship. It isn't even necessary. Even if it were, on the basis of far less information you have provided at least three disabilities on presumptive cases in the past.

Now, what other information do you have on—let's take, for example, the cardiovascular disease—that would give you cause to provide that compensation on presumptive disability?

Miss STARBUCK. I am going to let Dr. Custis speak to that. But the Congress mandated to the Veterans' Administration a study which would identify, if such existed, a causal relationship between certain amputations of lower extremities and cardiovascular diseases, a mortality study.

Mr. DASCHLE. Miss Starbuck, didn't they show——

Miss STARBUCK. Excuse me, excuse me——

Mr. DASCHLE. Didn't they show there was only one incidence——

Miss STARBUCK. Excuse me, sir. May I conclude my statement?

Mr. DASCHLE. I think you need to clarify it before you move on.

Miss STARBUCK. Let me say why they did this. Because there had been in the past other studies which were in disagreement, one with the other, some saying no, there was no relationship, and the others saying yes, there was.

In order to bring the question to rest, the Veterans' Administration was legislatively mandated to conduct such a study. It was conducted by the National Academy of Science. It was reviewed and the conclusions were accepted, that cardiovascular disabilities were a secondary effect of the amputations of lower extremities, either doubly above the ankle or singly above the knee. There was no presumption about it. It was a conclusion that the cardiovascular disability was a secondary disability to the amputations.

Mr. DASCHLE. Well, I beg to differ with you, Miss Starbuck, because I think what the study showed was that there was a higher incidence, and if I read that study as you read it, they could not show that there was a causal relationship. And even after they failed to show that, there still is a great deal of debate within the scientific community on whether or not you can show any kind of a relationship at all. I find it incredible that on the basis of that one study, given the fact that there is a tremendous amount of disagreement within the community, that you could give that same kind of a compensation increase on the basis of a presumption.

Now, I applaud you for doing it, frankly. But what I am pointing out is that it is incredibly obvious to me that there is a double standard here that you can't address. Really, I don't care how much you can tell me about the information that you will cite as reason not to provide a presumptive disability, when on the other hand there is overwhelming evidence on the other side.

I would just ask one final question—I know I'm out of time—but I think it sums it up, the whole question of—if this glass was filled with water, and we put a tablespoon of dioxin in this glass, would you drink it?

Ms. STARBUCK. No, I don't think I would.

Mr. DASCHLE. Thank you.

Thank you, Mr. Chairman.

Dr. CUSTIS. I understand there are some Ranch Handers that would.

Mr. APPLEGATE. The gentleman from Mississippi.

Mr. MONTGOMERY. Thank you, Mr. Chairman.

I really disagree with my good friend, Mr. Daschle. I think it is a very well-prepared statement. Quite frankly, it is one of the best statements I have seen the VA come up with. It has a lot of complete data.

I welcome the witnesses this morning. I want to commend Dr. Custis for being appointed by President Reagan to serve 2 more years as the director of our medical facilities of the VA.

I would like to point out to the committee what Dr. Custis said, that they and the other members of the Veterans' Administration are working for the veteran and are certainly not trying to work against them. Having said that—and I will stay within my five minutes—Dr. Young, I know you're a Ph.D. Why don't you tell us something about what you have heard earlier today and what you feel about the testimony we have had. You're one of the most knowledgeable persons—I recall your testimony several months ago pertaining to this same subject.

Dr. YOUNG. One of the things we have been attempting to do in the past few years is to collect the world's scientific literature on the subject area. Indeed, in our collection, we maintain over 5,000 articles on the phenoxy herbicides and dioxins. We have, on occasion, and we will be very soon putting another contract out to do another scientific review of that literature. As you know, the Veterans' Administration paid for, under contract, an exhaustive examination of the world's literature, and that was published in 1981. Those data have been discussed before.

I would just point out that recently an international symposium was held where some 250 scientists from throughout the world gathered to discuss the issues of dioxin and human health, environmental fate, and some of the problems related to toxicology.

That volume was released 2 weeks ago, and I bring a copy for you today. I just want to show you how selective the literature can be used—and I think it is important to be aware of how selective literature can be used.

Our concern is that it not be selectively used, but that it be looked at in its completeness. That's where we're coming from I think on this issue. I would just point out the statement earlier by one of the witnesses with respect to the Dow reproductive study. In this new volume, the Dow reproductive study is, in fact, provided in its completeness. What our witness failed to take into consideration this morning was, in fact, that data that were available. He simply excluded, selectively, some of the information in that report. Thus, he compared 737 exposed conceptuses to 2,031.

It was a very interesting use of the data, but when you use the data as they are actually presented, their conclusion is that there were no statistically significant differences in crude or adjusted risk estimates for any of the pregnancy outcome in this study. There were no significant trends overall with increased duration of exposure. In conclusion, under the conditions of the study, there were no biologically meaningful associations between adverse pregnancy outcomes and paternal dioxin exposure.

I would give you this article to be included for the record.[1]

Likewise, on the issue of the industrial findings of an abnormal number of cancers. Indeed, the Monsanto work is also reported in this brandnew publication, and it is thoroughly presented with respect to not only the earlier work, the work of the individuals who had chloracne, but in addition, it includes the entire Monsanto population at Nitro, W. Va. I would just point out that their results are very interesting.

Quoting from the scientific article:

> It is interesting to compare the results of this study of Nitro plant workers potentially exposed to TCDD with the results of the study of Nitro workers involved in the 1949 TCP accident. The workers involved in that incident had presumed TCDD exposure as evidenced by chlroacne. The results of the two studies are similar, in that neither show an excess in deaths from any site of malignant neoplasms.

The recent study mentioned earlier today, the Dow study, found no excesses in total mortality or in deaths from malignant neoplasms among workers exposed to 2,4,5-T. These workers were probably exposed to very low levels of TCDD since no cases of chloracne were observed. Other studies of workers who developed chloracne resulting from TCDD exposures have been conducted and have been reviewed. At the present time, data from these various studies do not constitute corroborative evidence of a cancer risk to man or any particular cancer site.

That is just one example of what, in fact, the scientific literature does say. If you want to misuse it, you can, in fact, do so very easily. That's the problem with the scientific data. It can be miscited. But when it is considered in toto, a different picture begins to appear than what we have heard this morning.

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[1] See p. 25.

Mr. MONTGOMERY. Thank you, Dr. Young. I am sure the chairman would let some staff personnel, after the Members have had other questions, follow up on whatever else Dr. Young might have to say.

Thank you, Mr. Chairman.

Mr. APPLEGATE. Thank you.

Mr. Edgar.

Mr. EDGAR. Dr. Young, to follow up to the chairman's question and your response, are you suggesting that the administration is wasting money and is acting irresponsibly as it removes families from Times Beach, Mo. The very same literature that you're quoting could be used in testimony to indicate that the administration's policy toward removing people from potential dangers of dioxin exposure as related in Times Beach was an irresponsible action.

What is your opinion of that?

Dr. YOUNG. We have talked about the issue of acute versus chronic, of course——

Mr. EDGAR. No, I would like a specific answer. Doesn't the——

Dr. YOUNG. I am trying to give you that.

Mr. EDGAR. Doesn't the analysis that you have just given lend credence to the fact that the administration must obviously be acting inappropriately as relates to Times Beach?

Dr. YOUNG. It is the acute issue that was at stake in Times Beach.

Mr. EDGAR. The issue at Times Beach was exposure. It was an acute problem, of course. But we're talking about an acute problem in the lives of people who are impacted by an equal amount of exposure. The same kind of chemicals that were found at Times Beach have been found to be used in Vietnam.

Dr. YOUNG. Well, is it, in fact, an equal amount? I think that is a very important point we're trying to make. Is it equal? No, it is not equal. The chemical——

Mr. EDGAR. That's not the question. The question——

Dr. YOUNG. The chemical sprayed in Times Beach was very, very laced with dioxin.

Mr. EDGAR. The question isn't the amount of chemical used. The question is exposure. We have gotten witness after witness, even those who oppose giving any compensation, who have indicated that exposure to just the smallest amounts of this very toxic chemical, described by one of our earlier witnesses at a previous hearing as the most toxic substance known to human life, and your raising the question of level I'm not sure is accurate at this point.

Dr. YOUNG. Well, you keep twisting it around. Let's take another try.

Mr. EDGAR. Thank you.

Dr. YOUNG. In fact, in the situation in Times Beach, where we don't know all the consequences of exposure through that environmental situation there——

Mr. EDGAR. But we presume——

Dr. YOUNG [continuing]. The fear of little children going out and crawling on soil and eating that soil. You see, this was a continual situation for them. They were out there being exposed continually. It was the fear of not knowing what that continual exposure might——

Mr. EDGAR. What about the veteran from my district who used——

Dr. YOUNG. That is why they removed the folks from Times Beach. It was the fear of not knowing exactly what was happening. The veterans of the Vietnam conflict, they had returned. They were no longer being exposed in Vietnam.

Mr. EDGAR. You have justified the reason why I have cosponsored Congressman Daschle's bill, and that is the fear of exposure, the veteran in my district who cut in half one of the barrels used to transport the liquid used in the defoliation of Vietnam as a kabachi and using that he was quite exposed to the chemicals that were involved in that barrel. I would suggest that some of that exposure is the same.

Let's not argue that point. Let me ask you, given your expertise, can you state for the record who it is that you work for?

Dr. YOUNG. I work for the Veterans' Administration, assigned on detail from the Department of Defense.

Mr. EDGAR. So you are intimately aware of this issue as it relates to the Department of Defense as well as to the Veterans' Administration?

Dr. YOUNG. I have worked with herbicide orange since 1968.

Mr. EDGAR. Can you answer a question that comes up often and troubles me. We heard earlier a concern about the broadness of the legislation in relationship to Southeast Asia.

Did the Army and Air Force use this defoliant around air bases in Laos and Cambodia as well as Vietnam?

Dr. YOUNG. The records are not clear as to that particular issue. The possibility exists that, indeed, the material could have been used. It is unfortunate that the records are not clear as to whether they were. In fact, we know in the situation in Thailand there was some use around one of the bases. Whether it was agent orange, we don't know.

Mr. EDGAR. Do you know which base it was?

Dr. YOUNG. No, but I can provide that to you.

[Subsequently, the Veterans' Administration furnished the following information:]

The defoliation test program was conducted on lands of the Replacement Training Center of the Royal Thai Army (Khai Thanarat) southwest of Hua Hin in northern Prachaub Khiri Khan Province on the Kra Peninsula. The two test sites were located at a latitude of 12 degrees 20 minutes north, and a longtitude of 99 degrees 50 minutes east, approximately 21 miles (35 km) from Hua Hin.

Two areas herein called Test Sites I and II were delineated by the Commanding Officer of the Royal Thai Army Replacement Training Center for aerial applications of chemicals in the test program. Total treatment areas of 1,400 acres (3,500 rai) and 2,000 acres (5,000 rai) were allocated for Test Sites I and II, respectively.

Mr. EDGAR. Do you know if similar use was made along the Ho Chi Minh Trail as well as in the Philippines?

Dr. YOUNG. As well as in the Philippines?

Mr. EDGAR. Yes.

Dr. YOUNG. The phenoxy herbicides, formulated as agent orange, have been used at military installations throughout the world, including probably 200 military installations in the United States. If you served at Eglin Air Force Base during the Vietnam conflict, you could have been as massively exposed to the herbicides used there as you could have been elsewhere.

Mr. EDGAR. Would you have records as to airplanes that would take off from an air base, fly someplace, drop the chemical, and fly back again?

Dr. YOUNG. The HERBS tape is the record that we have, as complete as it is.

Mr. EDGAR. Would there be records to show that that chemical was dropped along the Ho Chi Minh Trail?

Dr. YOUNG. Of course.

Mr. EDGAR. In the maps that we were provided, we don't see that.

Dr. YOUNG. Well, it depends on what part of the Ho Chi Minh Trail you are looking at. It's a very long trail.

Mr. EDGAR. But in Laos?

Dr. YOUNG. There were missions in Laos, yes. They are recorded on the HERBS tape.

Mr. EDGAR. The information we get from the Defense Department is that it was used almost solely in South Vietnam and in the Vietnam theater, and when we ask questions specifically about Cambodia, Laos, Thailand, the full extent of that area, we get the answer that you gave, in terms that we don't have complete records. It would seem to me that we would know where it went.

Is that possible to provide for the record?

Dr. YOUNG. The HERBS tape has been provided for the record before.

Mr. EDGAR. In Laos and Thailand, as well as Vietnam?

Dr. YOUNG. Well, the completeness of the HERBS tape, the data are there. Mr. Christian of the Army Agent Orange Task Force would be a better individual to seek that particular data from.

Mr. EDGAR. I have additional questions but will wait for the second round.

Mr. APPLEGATE. Thank you, Mr. Edgar.

Mr. Daschle.

Mr. DASCHLE. Mr. Chairman, I would like to pursue a couple of questions.

First of all, I am not clear, Dr. Young, on your statement with regard to Laos. Are you saying there was no spraying in Laos?

Dr. YOUNG. We know there was spraying at the border between Vietnam and Laos, yes.

Mr. DASCHLE. Yes, because I just sent my staff for the information, that we have ample information that there was all kinds of spraying—here he comes with the data now—on the exact areas with regard to spraying, and the fact that there was quite a bit of spraying in Laos. So we want to make sure the record is clear on that point.

We were talking earlier about your expertise, and I know that you are quite an expert in much of this area. You have done a lot of your academic work in this area.

You have a doctorate degree, is that correct?

Dr. YOUNG. That is correct.

Mr. DASCHLE. What was your thesis in regard to getting your doctorate degree? What was the title of your thesis?

Dr. YOUNG. It was on both 2,4-D and picloram.

Mr. DASCHLE. What was the title of your thesis?

Dr. **Young**. Well, that's a long time ago. It had to do with the sensitivity of various systems, plant systems, and its persistence——

Mr. **Daschle**. Certainly you remember the title of your thesis, come on.

Dr. **Young**. It was a very long thesis, and in my case it has been many years.

Mr. **Daschle**. Well, paraphrase it for me.

Dr. **Young**. It had to do with the diurnal regimes—the title actually included the diurnal regimes, the impact of Tordon or picloram, and 2,4–D on the plant systems and its persistence in the environment.

Mr. **Daschle**. Was the essence of your title and the thrust of your thesis that you could use these chemicals without any harmful effects?

Dr. **Young**. Well, no.

Mr. **Daschle**. That is certainly the impression I had from the title. But could you provide the committee with the title of your thesis?

Dr. **Young**. I would be pleased to.[1]

Mr. **Daschle**. I would appreciate it. I think that is an important point.

I think we have also established some other important matters here that we really need to clarify. First of all, that the law requires a manifestation of disability, not causal effect. I think that is the point that is totally missed in your statement. You lay out the entire statement trying to make a case that there is no causal effect, and yet that isn't what the law requires, a causal effect.

We have already demonstrated, too, that there are at least three diseases—and there may be others—cardiovascular disease, diabetes, and MS, in which there can be shown no causal effect, and yet that is exactly what we have decided in compensation, that there is enough of a presumptive disability here to provide for compensation.

So I think that we have seen a very blatant double standard here in your attitude toward this legislation, and I am very hopeful that at some point you can come to the conclusion with regard to dioxin that you already have with other diseases.

Another Federal agency disagrees with you vehemently. The EPA has made it very clear that it is perhaps the most poisonous synthetic chemical known to man. I'm reading right from their own document. They go through specific diseases, including disfiguring, multiple lesions, liver dysfunctions, involuntary rapid movement of the eyeball, showing all kinds of specific diseases.

You know, I think we have got the Federal Government even at odds with itself, and yet that one agency, who has the wherewithal to provide for compensation based on what findings we have within the Federal Government today, we have opposition. I am really kind of disappointed in that and I am hopeful we can send a message——

Dr. **Custis**. May I respond?

Mr. **Daschle**. Yes.

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[1] See p. 432.

Dr. **Custis**. We do not disagree. This is, indeed, a highly toxic agent. The manifestations that you just quoted coming from the EPA are acute toxic manifestations. The problem is, which are latent, chronic——

Mr. **Daschle**. Oh, come on, Dr. Custis, don't give me that.

Dr. **Custis**. I give you exactly that.

Mr. **Daschle**. No, you can't give me that because you haven't established that same kind of procedure for other presumptive disabilities. How can you say on the one hand it has to be that way for agent orange, and on the other hand you are willing to forgive the lack of information.

Mr. **Murphy**. Mr. Daschle, what we are saying is entirely consistent. I think I have to point out——

Mr. **Daschle**. It isn't consistent. It's a double standard.

Mr. **Murphy**. The law is the relationship to the period of military service, the manifestation during the period, or a connection with that period.

Mr. **Daschle**. You can't show that——

Mr. **Murphy**. If it was manifestation alone, a manifestation subsequent to service could not be service connected.

Mr. **Daschle**. That's not true. Tell me again how a veteran, after 7 years, can come into the VA and say "Now I have MS and I want presumptive disability". Where is the manifestation in his record that he had any kind of signs of MS back then?

Mr. **Murphy**. That is the nature of MS, as Congress decided. Congress told us that. Because that is the nature of MS, it is not necessarily the nature of everything else.

Mr. **Daschle**. The point I am making is that we have already established a tremendous precedent here. We haven't failed to follow through with that precedent when we talk about exposure to agent orange. It's a double standard, as clear as the nose on your face.

Mr. **Murphy**. All I can say is your basis for saying we have already established a precedent I do not agree with. I do not believe that is an accurate statement.

Mr. **Daschle**. Well, forgive my outburst.

Mr. **Edgar**. Would the gentleman yield?

Mr. **Daschle**. Yes.

Mr. **Edgar**. Mr. Murphy, you made a real strong case, then. If we pass the bill, then you, as a staff person and legal person for the VA, would come up next year and testify that because Congress determined that these particular diseases were due compensation as a service-related disease, you would defend that in the same way you have just defended MS?

Mr. **Murphy**. No. It is my understanding that Congress' determination with regard to MS was based on medical knowledge about the disease itself. If you enact this bill——

Mr. **Edgar**. Well, aren't we basing—you know, we differ in terms of the literature, but aren't we basing it on the same kind of medical information?

Mr. **Murphy**. I don't believe that is the case, Mr. Edgar.

In this case, as I understand it, you would distinguish MS in that it is medically known and——

Mr. **Daschle**. Would the gentleman yield, only to make a point? I just can't help it, but I have to say it again. There isn't any consensus of information on—If you look at the documents on either side of MS, you're going to find there is a balance there, probably a far greater imbalance, in fact, on the other side against MS. But nonetheless, we have already made that——

Dr. CUSTIS. Mr. Daschle, there is, indeed, a consensus regarding the course of multiple sclerosis. It is an extremely slow, progressive disease.

Mr. DASCHLE. Dr. Custis, I don't deny that. But you tell me about the causal relationship. There is a big difference.

Dr. CUSTIS. Let me finish, please.

The cause is not known, but the course is thoroughly understood. The consensus is established, that it takes years and years for the course of multiple sclerosis to evolve. The key here is, did it start during active duty? Did a soft tissue sarcoma, whether it is due to dioxin or vinyl chloride or what the devil it is due to, did it start during active duty? That is what we're after.

And while I have my mike, may I say to the distinguished Congressman from Philadelphia that I am flattered by his quoting me, but I would hope the record would put the quotation marks in proper position. The latter part of your statement was yours. The first part of your statement was mine.

Mr. EDGAR. That's correct. The record will show that distinctly. Do you want me to read it again?

Dr. CUSTIS. No, sir. [Laughter.]

Mr. APPLEGATE. Thank you. Mr. Edgar?

Mr. EDGAR. Are you done?

Mr. DASCHLE. For now.

Mr. APPLEGATE. Five minute rule, gentlemen.

Mr. EDGAR. Dr. Custis, first a compliment. The last 2 days we spent in three hospitals in Pittsburgh, one hospital in Erie, and one hospital in Butler, with a large number of members of the Hospital Subcommittee. We had, in fact, 12 members at a hearing on Monday of this week, where the regional director spoke in Pittsburgh, and then we had the opportunity to go on foot to as many pharmacies and clinics and research labs and acute care facilities and long-term facilities as we could find in western Pennsylvania.

I have to say that, to a person, the medical staff and personnel that we met were super courteous, capable people. We asked them issues relating to agent orange and also issues related to the aging veteran, in terms of the quality and quantity of staffing requirements in each of the facilities, and I think we brought back some information that we want to share with you about the facilities that we visited. In general, we found them to be very well providing care and services to the veterans, given the limits of funding that we have.

That was the first of six regional hearings that we plan to have across the country and we invite you and your staff to participate in those hearings, to walk with us, to go with us if you wish, and we will try to keep you informed on those opportunities. Particularly helpful I think were the freshmen Members who were there, because for some of them it was the first time they had a chance to see the long-term care facilities, the chronic care facilities, the acute care facilities, the ambulatory care facilities that we provide.

I think the schedule helped us to see a variety of the contexts under which your service operates.

I kind of enjoyed the arguments and fights that we just had, and I hope that as you come with fear and trembling you will understand that we are both trying to understand and pursue these things, and I appreciate your point about partial information and partial knowledge.

The reverse is also true. You can get flooded with a variety of statements if you just weigh the literature and pile literature on people's desks and not read the literature. There may be 50 papers that all say one thing and one thoughtful paper that actually gets to the heart of things. There is a great difference of opinion among the staff of the VA Committee here. I see heads shaking behind you when questions are asked, that opinions have already been formed.

I think the Congressman from South Dakota and other Members of Congress have come to the conclusion that by the time we wait for all of the definitive answers that you may wish to have to make the decisions, that many of us may not be in Congress any longer, many people who may have been impacted by the defoliants, if, in fact, there is that causal relationship between the use of the defoliant, exposure and systemic diseases, that when we actually find that definitive information it may be too late.

You pointed out very correctly that with MS there is some information, a body of information, that talks about the gradual nature of it, the slow and insidious way in which MS confronts people. I, frankly, have the same fears about excessive exposure to dioxin, and that is one of the reasons I took on Dr. Young on his great defense on how the literature doesn't show the impact, because that same defense was used by people who opposed the EPA's decision several times, most recently in terms of Times Beach. So while you may agree that extensive exposure to dioxin does have more causal relation than the slow or smaller exposure, there are some real questions and questions that we need to thoroughly examine.

Have you had a chance in central office to take all of the reports from the 172 hospitals and the 200 and some odd ambulatory care clinics that have done thousands of examinations of agent orange in your hospital system, have you taken that material and computerized it at all and looked at any trends? Do you have any information based on the material you are getting into the central office?

Dr. CUSTIS. Yes, sir; we have. Could I defer to the vice president in charge of the registry?

Mr. EDGAR. Why don't you state your name for the record.

Dr. SHEPARD. Yes. I am Dr. Barclay Shepard, Acting Director of the VA's Agent Orange Project Office.

In answer to your question, Mr. Edgar, we are in the process of analyzing the data which have come from some 106,000 examinations that we have done in the agent orange registry process.

Unfortunately, when that process was first put together, we did not collect specific medical information. The purpose of that registry was more to identify Vietnam veterans who are concerned about the possible health effects, not designed specifically to get specific medical information. We have corrected that deficit in that we have revised the coding information, the computerizing process.

However, we have looked back at some particular illnesses, specifically, fairly recently, the matter of soft tissue sarcomas. We have identified in the agent orange registry some 1,000 cases of malignant neoplasms. Of those, we have preliminary information that 14 are in the group known as soft tissue sarcoma. We are now looking at identifying specifically what kinds of soft tissue sarcomas there are in order to determine whether or not there is a particular type of soft tissue sarcoma which is appearing in any unusual pattern as compared to what would be expected in the general population.

What we have done is analyzed the proportion of soft tissue sarcomas in the total numbers of malignant cases in the registry, and that proportion is essentially the same as one would find in that same kind of proportion looking at the number of malignancies in the general population. So we have made that kind of preliminary analysis.

We are continuing to gather other information and will report to this committee as it develops.

We are doing a number of other studies relating to the soft tissue sarcoma issue. Quite recently we have developed a protocol working with the Armed Forces Institute of Pathology, which has the largest soft tissue sarcoma registry in the world. We will be working with them on a case control study, looking at the risk of Vietnam veterans developing this particular group of tumors.

A number of other studies are going on around the country. The National Cancer Institute has initiated a study in the State of Kansas. The NIH has provided a grant for a soft tissue sarcoma study in the State of Washington, and there are a number of other studies related to this issue going on both in this country and around the world.

Mr. EDGAR. Are the tests now given to veterans who come in for agent orange examinations consistent throughout the country, in terms of the questions asked?

Dr. SHEPARD. Well, as you may know, Mr. Edgar, when an individual comes in for a health examination, there is a standard set of questions that are asked. But as these questions are answered and the answers point to illnesses of one kind or another, or symptoms of one kind or another, then the emphasis of the questioning may shift to evaluating what specific complaints are being presented. So although it starts out as a standard general procedure it may later focus on that particular veteran's complaint or illness.

The physical examination is essentially a standard physical examination that one would provide——

Mr. EDGAR. How do you code the information if all the information is different?

Dr. CUSTIS. I suggest, Mr. Edgar, if we could supply for the record the March 1, 1983, last version, Circular 10-83-38, it gives specific descriptions of that coding.[1]

Mr. EDGAR. That would be helpful.

Do you test for porphyrins?

Dr. SHEPARD. Not routinely.

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[1] See p. 433.

Mr. **Edgar**. Is there any evidence that testing for porphyrins may give you a better handle on their role in this area of exposure?

Dr. **Shepard**. There are a number of minor variations from the norm on porphyrins. There is nothing very specific about most porphyrin derrangements. Therefore, the standard testing for porphyrins would not point to a particular relationship with the herbicide. When indicated, as the questionnaire is taken and the physical examination is conducted, if there seems to be an illness suggested by either the questionnaire or physical examination, then porphyrins are ordered to elucidate and shed further light on whatever illness might be present.

Mr. **Edgar**. Thank you very much.

No further questions, Mr. Chairman.

Mr. **Applegate**. Thank you, Mr. Edgar.

Does counsel have any questions?

Mr. **Fleming**. Thank you, Mr. Chairman.

Several questions have been answered that the chairman wanted me to ask in his absence, but I do have two or three the chairman wanted to ask for the record.

I think it is fair to say, as Mr. Edgar suggested, that there is a difference of opinion among some members of the committee on this issue. The chairman would like to get your response to some of the comments made by some of the witnesses earlier this morning.

What are the limitations inherent in drawing from the animal studies conclusions regarding the health effects in humans from dioxin?

Miss **Starbuck**. I would ask Dr. Shepard to answer that.

Dr. **Shepard**. I think that in talking about animal studies and dioxin and extrapolating that information to humans, one has to bear in mind that there is a wide species variability as to how certain species react to dioxin. For example, the amount of dioxin it takes to kill a hamster is about 5,000 times the amount that it takes to kill a guinea pig. The hamster is much more resistent than a guinea pig, about 5,000 times.

Now, here are two relatively closely related rodent species, so it is difficult to choose one animal or even a group of animals where the species variability is so wide and use that information to extrapolate to the human.

What we do know about humans has resulted largely in the workplace and in industrial accidents such as occurred at the Monsanto plant in Nitro, W. Va., and at Seveso, Italy. In those observations, it appears that humans seem to be relatively more resistent than some animals. For example, in the Seveso accident there were a number of wild and domestic animals that were acutely killed by the exposure to this rather heavy concentration of TCDD. To our knowledge, there has been no acute human death as a result of TCDD exposure.

Mr. **Fleming**. What do you consider to be the limitations of the Swedish studies in terms of their validity for proving cause and effect?

A couple of witnesses have placed great weight on the Swedish study.

Dr. **Custis**. I think the consultant that served OTA very well, Dr. Remington, identified the reservation in the study that other critics later on repeated, namely, the apparent deficiency drawback of a case/control technique, the inherent bias of such a technique as compared, for example, to the cohort technique. The reputation of Dr. Axelson and Hardell were excellent, as Dr. Remington points out, and they did, indeed, have a very careful control of their study. But the drawbacks of the case/control technique are where, under peer review, it falls down.

I ran across what I thought was an excellent example of the problem, an illustration of how case/control studies can be misleading, if I can rapidly lay my hands on it. Well, as best I can recall, it goes like this:

In the mid-seventies, on the basis of a case/control study, there was found to be an increased incidence of breast cancer in women who had been on reserpine for hypertension. It became a national issue. It was in the lay press. There was a fair amount of concern, if not hysteria, over the drug reserpine. It precipitated multiple studies, more carefully controlled studies, cohort studies, and over a period of about 2 or 3 years that whole scare was laid to rest. That is what can happen in the case/control technique that is not properly followed by more careful procedures.

Mr. **Fleming**. Well, testimony was presented yesterday, Dr. Custis, that indicated that the Air Force ranch hand personnel may not have received the same level of exposure to agent orange as our ground troops did, thus casting some doubt on the validity of the findings that may result from that study.

Could you comment on that?

Dr. **Custis**. We had that very question under discussion yesterday when we were brainstorming together. I think Dr. Young responded in the most [illegible] way, so I would defer to him.

Dr. **Young**. The [illegible] study not only includes the pilots that flew the C–123's, [illegible] includes the men that handled the herbicides on the ground, [illegible] that did the repairing of those aircraft that were saturated [illegible] the herbicide.

We have heard the [illegible] the ranch handers would go out, fly for a little while [illegible] herbicides, come back and go take a shower. I think that is clearly not true. You also must remember that they went out and sprayed every day. Many ranch handers were involved in over 400 missions over a period of 1 year. We have photographs showing them dripping with herbicide.

I am sure they, in fact, when they had the opportunity, did go take showers. But clearly the information that the Air Force has been gathering on the exposure of the ranch handers shows that many of them were massively covered, for sometimes days on end. Many ranch handers wrote home asking their wives to send them additional boots because the boots they were using were rotting away, because they were standing in herbicide all the time.

The data we have on ranch handers indicates that if you talk about a dose response relationship, they were heavily exposed. The results of that epidemiologic study should be very valuable, if you're talking about exposure to agent orange, to the herbicides.

Mr. **Daschle**. You really didn't answer the question in regard to bathing and cleansing themselves on a daily basis. But could you also address the other scientific question raised so often challenging the credibility, that the size of the sample is going to be very small and from that small sample it is going to be very hard to conclude anything.

Would you address that?

Dr. YOUNG. Yes. One of the fears that we did have was the participation, that the sample would not be large enough to do much with. But that didn't come to pass. It was the fear that the National Academy of Science had, that the sample size would be too small, participation would be too small. But, in fact, what has happened is that we have a tremendous participation by the ranch handers.

The controls, the very carefully matched controls, that's the key here. Not only do you have 1,200 ranch handers that came out to participate, but you also had individuals who were the same age, plus or minus a few months, who served in Vietnam at the same time, who had the same sort of jobs, who were of the same race. That's what makes the study much more powerful than it has been recognized by a lot of the critics.

Mr. DASCHLE. Well, the National Research Council is very clear. It says the study lacks statistical power to uncover any effect as to the common disorders mentioned in the complaints of veterans. In summary, the National Research Council says here the overall study, including the morbidity and mortality components, is seriously limited by the insuffficent sample size.

Dr. YOUNG. Please go ahead and cite now, in the same document, the minority report by Dr. Kurland. Of course. You see, the Committee on Toxicology was the one selected to review it, not the Committee on Epidemiology. The epidemiologist, Dr. Leonard Kurland, Mayo Clinic, Rochester, Minn., wrote the minority report that indicated the study was a very powerful and well designed study.

Mr. DASCHLE. Well, there is no question that it's a powerful study in the sense that it is going to be helpful.

Dr. YOUNG. It's statistically powerful.

Mr. DASCHLE. No. The majority of people representing the National Research Council said it was totally worthless with regard to any firm conclusions because of the size. I think that needs to be stated for the record.

Dr. YOUNG. Well, Dr. Lathrop and the team who are conducting the study, and the oversight committee of distinguished scientists who are reviewing the ranch hand work, I am sure will have a chance to present that information at a later date.

There is a point to be made here, too. That particular body, the National Academy of Sciences, was one of the only bodies that did have a criticism. If you look at what the Armed Forces Epidemiology Board said about the ranch hand study, they had high praise for that study. The Air Force Scientific Advisory Board had high praise for that study. Those are scientists not within the Air Force but selected from within the scientific community.

Mr. DASCHLE. Would you call them independent?

Dr. YOUNG. Oh, yes, I would. Sure.

Mr. DASCHLE. Your view of independence and mine is a little different.

Dr. YOUNG. Because a scientist works for a Government agency, or is hired to review a document, doesn't mean that that scientist is going to compromise his integrity. That's a terrible implication you are making.

Mr. APPLEGATE. Mack, do you have further questions?

Mr. FLEMING. I have one additional question—I have others, but I guess we're pressed for time and I will submit those to be answered in writing.

The final question I have relates to a question asked by a witness this morning. I believe the physician from the South Dakota hospital, or possibly Dr. Epstein, asked: "What are we waiting for?" We would like to have your comments as to what the agency is waiting for.

I realize the individual asking the question is not an adjudicator. So, from the standpoint of adjudication of claims, what would the VA be waiting for?

Miss STARBUCK. I think the VA is waiting, Mr. Fleming, for a consensus to be reached in the medical community, as Dr. Custis earlier stated it, that would provide to us a solid reason, at this time departed from Vietnam, to relate a disability to military service in Vietnam.

Mr. FLEMING. Thank you, Mr. Chairman.

The Chairman of the full committee has additional questions, but with the agreement of the committee I will submit those in writing to be answered by the agency.

Mr. APPLEGATE. Absolutely.

Counsel on the minority side.

Mr. WILSON. There may be some questions, Mr. Chairman, submitted for the record by the members on the minority side of the committee. I would like to have unanimous consent to put those in the record.

Mr. APPLEGATE. Without objection, that shall be done.

Mr. WILSON. Just two quick questions, Mr. Chairman.

Dr. Custis, I thought you were about to respond a while ago, and while I hate to open the question again on the circulatory matter, were you about to respond on that?

Dr. CUSTIS. I would like to add something on that, Mr. Wilson.

The congressional publication of the 96th Congress, in which the Administrator of the Veteran's Administration submitted all studies to the Congress, references some 13 studies in all that antedated the congressionally mandated study that was then done by the National Academy of Science. The 13 research projects that had antedated that gave differing results.

I think what we had here was a situation very similar to where we stand as regards soft tissue sarcoma, where there has been introduced, under the stimulus of the Swedish studies, a high level of concern and controversy. There is a great need to now go on with an epidemiologic study that will hopefully straighten things out and provide the evidence that will stand up under peer review and will lead to a consensus. It was just such a study that was conducted by the National Academy that became the reason for a resolution of all the preceding argument based on previous studies and led the VA to take a stand finally, satisfied that a consensus had been arrived at, that consensus being that there was, indeed, a higher incidence of cardiovascular disease in above-the-knee amputees, single or bilateral.

These studies are still going on. For example, the VA in the Bronx, I believe, or Brooklyn.

Mr. WILSON. Doctor, what is the title of that report?

Miss STARBUCK. It is Senate Committee Print No. 6 of the 96th Congress, 1st session. It is dated February 15, 1979.

Dr. CUSTIS. The title being "Causal Relationship Between Service-Connected Amputation and Subsequent Cardiovascular Disorder—A Review of the Literature and a Statistical Analysis of the Relationship, Including the Results of the NAS Study."

Mr. WILSON. Thank you, Dr. Custis.

Dr. Shepard, on a number of occasions you have pointed out the distinction between being exposed to dioxin and being exposed to phenoxy herbicides. Would you, in lay terms, just elaborate on that briefly?

Dr. SHEPARD. Yes, sir. I think with the continued use of these terms it becomes tempting, or it inadvertently results in a mixture of the terms. I have heard even this morning people using the word "dioxin" to mean a herbicide. Dioxins are not herbicides. The phenoxy herbicides, 2,4-D and 2,4,5-T are the herbicides. Dioxin is contained in minute quantities in 2,4,5-T as a contaminant during the manufacturing process. But I think it is important, when you look at literature, when you look at reports, when you look at statements, to make sure that the distinction is made between the phenoxy herbicides and dioxins. They are not synonymous and the distinction should be very clearly made.

I might use an example of that. In the Administrator's testimony of yesterday, he spoke about the fact that there is scientific consensus on the matter of exposure to dioxins and the development of chloracne and porphyia curanea tarda. I think there were some suggestions made here that the Administrator has stated that there is a scientific consensus on the matter of these diseases relating to exposure. But the fact that it is dioxin and not the herbicides I think is the important issue to bear in mind.

Mr. WILSON. Thank you, Doctor. Just one final brief question, Mr. Chairman.

Miss Starbuck, have you ever seen a case where chloracne has been diagnosed—and I know your testimony alluded to this—and the veteran had been in Vietnam and you had conceded he had been exposed to agent orange when that case has not been service-connected?

Miss STARBUCK. No, sir, we have not.

Mr. WILSON. Thank you, Mr. Chairman.

Mr. APPLEGATE. Thank you very much, Miss Starbuck. We thank you and Mr. Murphy, Dr. Custis, and Dr. Young for coming in and appearing before us.

Miss STARBUCK. Thank you, Mr. Chairman.

Mr. APPLEGATE. Now we will get to our final witness, Dr. Peter Kahn. Dr. Kahn is with the department of biochemistry and microbiology at Rutgers University.

Dr. Kahn, we do have your statement which will be entered into the record. You may proceed as you wish. You may summarize, and we will, of course, have some questions for you.

## STATEMENT OF PETER C. KAHN, PH. D., ASSOCIATE PROFESSOR OF BIOCHEMISTRY, RUTGERS UNIVERSITY, APPEARING ON BEHALF OF THE NEW JERSEY STATE AGENT ORANGE COMMISSION, ACCOMPANIED BY WAYNE WILSON, EXECUTIVE DIRECTOR, NEW JERSEY STATE AGENT ORANGE COMMISSION

Mr. KAHN. In presenting the testimony that will follow, and in responding to your questions, I would like to make it clear that I am a research biochemist. I am not a physician. My training does overlap that of a physician to a considerable degree, but I wish that point to be made clear at the beginning.

I am going to read this, as it is rather brief, and then I will take questions. I will not spend nearly as much time as your preceding witness.

Thank you for the opportunity to appear here. My name is Peter Kahn. I am an associate professor of biochemistry at Rutgers, the State University of New Jersey, and I am also a member of the New Jersey Agent Orange Commission. With me today is Mr. Wayne Wilson, who is the executive director of the commission and who is a two-tour veteran of service in Vietnam, medically retired from the Army.

I appear here today on behalf of the commission, and we support H.R. 1961, and our arguments in its favor follow. The commission approved this testimony at its meeting of April 15, 1983, a week ago.

To convey some sense of the immediacy of the problems faced by Vietnam veterans who are ill, we offer two case histories from our files. Next, we suggest two conditions that we believe should be added to those now in the bill. We then discuss the use and misuse of science in formulating public policy and, lastly, we examine some of the moral and political issues that are involved.

Case one, whom I am calling Mr. A., was 37, married, with three children. He was a "river rat" in the Navy from 1965 through 1967 and was discharged in 1971. His work in Vietnam involved running small craft up and down the rivers of the Mekong Delta, and he worked on a barracks ship as well. He was decorated. The rivers of the Mekong, by the way, were heavily sprayed.

Until he fell ill in 1979, he earned a comfortable living as a master chef. His primary diagnosis was of cryoglobulinemia with associated vasculitis. The condition is an unusual immunological disorder—and I will have some comments about immunological problems later. Other problems developed as a consequence and the aggregate result was his death late last year.

His widow now works two jobs. That and social security are all they have. The family has large debts arising from the long illness. Their oldest child, a daughter, graduates high school this year. She is a good student and wants to go to college. The American Legion has established a scholarship fund in his memory with the stipulation that his children shall have first claim on it. It is small and may not be enough.

Case two, Mr. B., 36, married, with four children. He was an aircraft mechanic before entering the Army, served in Vietnam in 1968–69. He now earns his living as an auto mechanic. He did helicopter repair work in Vietnam and he was in combat.

The oldest of their children, a girl, was conceived before he went to Vietnam. In her mother's words, she has "no real health problems, has exceptionally high IQ; school psychological evaluation done; child extremely artistic, athletic, and a genius." Parental pride may be showing there. The other three children, all conceived since Vietnam, have problems. One has arthritis. Another is asthmatic. All suffer from frequent respiratory infections, including bronchitis and pneumonia. Two have been hospitalized. All three have required speech therapy, and there are other problems as well.

The financial burden of continuing care makes it necessary for Mrs. B. to work two jobs. In January of this year she contacted the commission. It is common for our first contact with veterans to be through their wives. The men often think it useless to seek outside help. We sent her a copy of our "Vietnam Veterans' Self Help Guide" which contains a brief questionnaire. Here is her reply, dated January 11, 1983—and where there are quotation marks in the text of her letter, they were in the original, and also the emphasis.

> Dear Commission: I finally succeeded in getting my husband to fill out the enclosed form from your "self-help guide." I have tried for years to get him to have a physical done to no avail. So finally he got so sick he asked me to make him an appointment, which I did, and on June 28, 1982, he had his physical done and many other tests. He came home from Vietnam with sores on his chest which have since enlarged and spread to other areas such as his back and shoulders. If I had known that he was eligible for a free exam at the V.A. hospital I would have pushed him to go sooner. It's only for this reason he never had a physical done—we couldn't afford it. We have very high medical expenses from our children's illnesses. We just couldn't put any more out.
>
> The people at the V.A. hospital in Delaware were nice, but boy it's a long wait. They would not commit themselves to say anything in regard to Agent Orange except that he has nine of the ten symptoms all the other boys have.
>
> My husband constantly has chest pains (sometimes worst than other times). Pains particularly around the main sore in the center of his chest (which by the way is three times bigger than it was when he came home from Nam with it). He has numbness in hands, arm, legs, breathing difficulties, inflammation/irritation of sores on chest, constant trouble with bowels—either constipation or diarrhea, very seldomly has normal bowel movements, occasional headaches, dizziness. He gets cold, loses feelings and becomes weak and pale looking. It frightens me to know and feel that something is wrong with him but yet all tests performed proved to be medically negative. In other words they can't medically find anything wrong. They also said (but it isn't in writing) that his appendix is enlarged "possibly" from chemical poisoning or "whatever".
>
> I myself don't understand any of this red tape medical talk, but being married to the same man for 15 years I can tell you something is wrong with him. When he first came home from Nam he dreamed bad dreams and many nights I would become a victim and find myself fighting him off, for he would physically fight me thinking I was Vietcong. I grew to understand and stood by him. He still dreams but it's not as bad as it first was when he came home.
>
> I can slowly see his health is tearing down and all this talk about Agent Orange I don't understand. Why would the U.S. Government spray such a deadly chemical over Nam knowing our boys could be harmed? I often wonder about that and now the Government doesn't want to recognize our men who fought hard in Vietnam, many who died, as veterans. So its after a great deal of persuasion getting him to fill out this form I'm finally mailing it to you. He knows about the form, but he does not know I have written this letter to you.
>
> He has another appointment tomorrow in Delaware for he still has pains in chest (in center of main sore), has numbness, weakness, and recently developed sores on his legs. His physical appearance is run down and pale looking. Yet they can't find anything wrong with him except for his exposure to chemicals in Vietnam.
>
> So tomorrow will be another long day of waiting so they can say "nothing" is wrong; but then I wonder. Only time will tell.

I'm not much at writing letters, so forgive the wording and penmanship. I hope further research would be proven to be beneficial for all boys who were exposed to the chemicals so they can be cured or helped any way possible. I wish there was something I could do for my husband—and I guess just being by him is enough for him.

I don't know if my letter is of any importance to you, but I felt I had to write.
Cordially, Mrs. B.

Mr. B. knows of his wife's letter now, and they agreed to let it be used here. Mrs. A. also agreed to our use of material from her husband's file.

The medical conditions which we suggest adding to the bill are melioidosis and the family of cancers called the malignant lymphomas, including the leukemias. Melioidosis is a bacterial disease. Its origin has nothing whatever to do with agent orange. The disease is endemic in Southeast Asia. It can take two forms—acute, which is often fatal, and chronic. Years can elapse between exposure to the bacterium and the onset of symptoms.

Cases were diagnosed in troops in the field during the war. Cases have arisen in Vietnam veterans since their return, sometimes triggered by physiological stresses such as influenza. The number of cases proven to have arisen from exposure in the Western Hemisphere can be counted on the fingers of one hand. The likelihood, therefore, of a Vietnam veteran's case of the disease having originated anywhere but in Southeast Asia is almost nil. The number of cases expected to arise in veterans is small.

The evidence supporting the inclusion of the malignant lymphomas is of four kinds:

One, Lennart Hardell and his associates in Sweden, the people whose work on soft tissue sarcomas led to the current interest in those cancers, have also studied the lymphomas. A preliminary case report appeared in The Lancet in 1979 and a detailed case/control study was published in The British Journal of Cancer in 1981. These results indicate that persons exposed to chlorophenols and phenoxy acids had a fivefold to sixfold greater incidence of these cancers than did unexposed persons.

Two, lumberjacks, occupationally exposed to phenoxy herbicides—this is work in this country—appear to have an increased incidence of lymphomas and leukemias.

Three, one study of American workers exposed to trichlorophenols found three deaths of lymphatic and hematopoietic malignancy when, for the size of the cohort, 0.88 were expected.

Four, dioxin is known to suppress the cell mediated immune response—remember that immunological problem in Veteran 1—in animals. Organ transplant patients, who receive immunosuppressive drugs for obvious reasons, have a greatly increased incidence of lymphomas.

The preliminary evidence is sufficiently suggestive, we believe, to justify at this time presumptive service connection pending further work. Although the research is by no means conclusive, we strongly recommend that in the context of the bill melioidosis and the lymphomas be included.

As a scientist, I can't tell you that the medical problems of Vietnam veterans are unequivocally the result of exposure to noxious materials during service there. I think that there is an abundance of circumstantial evidence to suggest such a connection, but that is not rigorous proof. The Federal Government—and you heard a good dose of it before I started—has generally taken the position that in the absence of such proof no action should be taken, and not only with regard to Vietnam veterans.

Political considerations sometimes alter that position, but they do so only occasionally, Times Beach being a case in point. The burden of justifying action is thus shifted to science. We are asked to produce evidence sufficient to "convict beyond reasonable doubt." Science cannot do that. There is alwavs reasonable doubt in the evaluation of research, particularly biological research. By requiring science to do what it cannot do before you, the Government, take action, you avoid taking action. In the process, science is discredited, and if that continues, the river of good that it has produced will run dry.

In shifting the burden on to science, moreover, you shift it away from its proper place, which is on you and on the rest of the Government. The issue before you is not primarily scientific; it is political and moral. There are no technological disasters. There are, instead, failures of political will and moral courage.

The families that we have described here today are in trouble, and they represent only a few of many in such situations. The problems of the men affect their families directly. If a man cannot keep a job, or if he has one but is so often ill that he exhausts his sick leave, he suffers doubly—from the illness and from the loss of self-respect that attends his inability to support his family.

How would any of us face the future with debilitating illness that doctors can neither diagnose clearly nor treat effectively? Feeling poorly, moreover, a man is less able to deal with the stresses that his frequent incapacity creates. Often his wife must work to make ends meet—both the cases I cited are of that kind—and the resulting fatigue reduces her ability to cope with the increased emotional needs of her husband and of the children. The children are not immune from the results. In addition to lack of money and continual uncertainty about the future, their parents frequently cannot provide the kind of predictable, secure family life that children need. The parents, moreover, usually recognize this, and the knowledge itself adds to their difficulties. Some marriages survive these stresses, and some do not.

We submit that the ultimate dollar cost to the country of not helping these men will far exceed the dollar cost even of generous help. Help, unfortunately, is an easily identified and vulnerable single target in a budget. The lack of help now, however, will grow into a multitude of items in many budgets—Federal, State, and local. Their connection to the subject of today's hearing will be obscured, but they will nonetheless be real.

In addition to direct dollar costs, moreover, there are other losses of material wealth, wealth not generated by people whose vigor is sapped and whose children's vigor is not nurtured as well as it could be. To all these a dollar value could be attached. Add to them the costs for which there can never be a money equivalent: damaged lives, anxiety, and, for some, dispair. We can write all this off as yet another cost of war, but if we do, we allow a rip in the fabric of a nation that can ill afford them to become a spreading hole. In the long run, we will pay handsomely for that.

If you pass this bill, then some of the people who receive help as a result may do so for illnesses that are not, in fact, service connected. If you vote it down, and if it is later shown that there is a connection between exposure to environmental hazards in Vietnam and later medical problems, then you will have declined to help men who have earned it at a time when it is needed.

We of the New Jersey Commission look at the same evidence that the Government finds inconclusive and find it suggestive of service connection, some of it strongly so. If we are right, and I believe that we are, inaction now means national trouble later, for in making a choice not to act, you raise questions in the mind of the people of the credibility of the Government.

Thank you.

[The prepared statement of Mr. Kahn appears on p. 454.]

Mr. DASCHLE [presiding]. Thank you, Mr. Kahn. That was a very moving statement. I think it certainly gives us another major incentive to want to act quickly on this legislation.

I think the thrust of what you have told this committee is a good message to end this hearing on. From what you tell me, and from what other witnesses have alluded to for the last couple of days, there will never be unanimity of opinion with regard to much of what we decide today.

Mr. KAHN. I think that is right.

Mr. DASCHLE. As you look at the bodies of information available and other diseases and their causal relationship to exposure, do you often or infrequently find a consensus of opinion within the scientific community?

Mr. KAHN. I have never found a consensus of opinion on anything completely. There is always somebody on the other side.

Mr. DASCHLE. So, in essence, you could say by continually seeking to find consensus, we will be simply on a track that is never ending?

Mr. KAHN. It's a smoke screen.

Mr. DASCHLE. So one could almost say that it may be just another way of opposing the legislation without outright saying so; is that it?

Mr. KAHN. Correct.

Could I make a comment about some prior testimony? Some noise was made about the selective use of scientific information, and reference was made in the case of soft tissue sarcoma to the number of studies that have come up negative. Specifically, there was a Finnish study of herbicide sprayers and a study of Swedish foresters, and there was some work in New Zealand.

I have here with me a review from the Lancet, from 1982, the May issue, by Coggon and Acheson, from the MRC—that's the Medical Research Council in England, the Environmental Epidemiology Unit. These are men who are commenting on Hardell's work in Sweden, only one paragraph and one sentence. These are the negative studies that are being taken up.

"No soft-tissue sarcomas were reported in a study of Finnish herbicide sprayers, but the number of men followed was small and a fivefold increase in risk might easily have been missed by chance——"

Mr. DASCHLE. A five-fold increase?

Mr. KAHN. Yes, might have been missed by chance, cited as evidence against soft tissue sarcoma.

"Similarly, the absence of any cases in a followup study of Swedish foresters and forestry foremen could well have been due to chance, since the expected number of soft tissue sarcomas was estimated to be only 0.1—" not 1, but 0.1.

"Again, there were no soft tissue sarcomas in a study of Swedish railroad workers exposed to phenoxy herbicides, but the total number of cancers found in these men was only 16." That was cancers of all kinds. The percentage of soft tissue sarcomas in the general population would indicate that you should not expect any.

Concerning the New Zealand result, I reviewed these studies I mentioned here myself. I have not seen the full text of the New Zealand study yet. However, in the comment here on the preliminary report—I will only read one sentence—"* * * but it is possible that in New Zealand a greater proportion of spraying has been from the air." In Sweden, most spraying was done from the ground using backpack and truck rigs and the exposure conditions would be quite different.

Mr. DASCHLE. Are you familiar with any reference to skin cancers in the Finnish report?

Mr. KAHN. No. I was in Finland this summer and I asked people about that. They weren't sure.

You may have this, if you wish.

Mr. DASCHLE. I think for the record that would be a very worthwhile document. If you would leave that with us, we would appreciate it.

[The information appears at p. 203.]

Mr. KAHN. It makes me angry when people talk about the use and misuse, the selective use of data, when they don't tell you this.

Mr. DASCHLE. It does me, too. I think it undermines unfairly the credibility really on both sides, and we don't want to do that. We want to offer as comprehensive a list of information as we can. Certainly you have done us another service in providing that for the record at this time.

Mr. KAHN. My files are open to the committee at your wish.

Mr. DASCHLE. Thank you.

I have no further questions. Mr. Chairman, do you have any additional questions?

Mr. APPLEGATE. Yes, thank you, Mr. Chairman.

Mr. Kahn, when was the use of agent orange terminated in Vietnam?

Mr. KAHN. In 1971. The median latency in soft tissue sarcoma and in the malignant lymphoma work is 15 years. That is the period between exposure and the manifestation of symptoms.

Mr. APPLEGATE. And when was the fighting terminated?

Mr. KAHN. In 1974.

Mr. APPLEGATE. Why did they terminate the use of agent orange in 1971?

Mr. KAHN. Largely because of political pressure here at home.

Mr. APPLEGATE. Is that the only reason? No scientific reasons?

Mr. KAHN. Oh, yes. The political pressure arose out of growing findings that there were dangers both to Vietnamese who were being exposed and to American troops. There were scattered reports in Vietnam at that time of an increase in stillbirths and deformities, and the AAAS, the American Association for the Advancement of Science, led a team out there. Owing to wartime disruption, they were only able to look at stillbirths. They couldn't look at malformation. They did find a suggestion of an increase but they couldn't do a complete study because of the disruption. So there were pieces of information beginning to appear.

In addition, the bionetics research laboratories published a report—I think it was in 1969, but my memory gets a little foggy—showing that 2,4,5-T, which at that time contained dioxin as a contaminant, when administered to laboratory animals produced an extraordinarily high incidence of stillbirths and malformed offspring. That was 2,4,5-T which contained dioxin.

That bionetics report really started the public ball rolling. Based on that, plus the findings from Vietnam, which were scattered at the time, plus the continued opposition of biological scientists here in this country to the use of chemicals in warfare, that combined to lead the Government to stop.

Mr. APPLEGATE. Thank you very much, Dr. Kahn. I appreciate your being here.

Mr. DASCHLE. Mack, do you have some questions?

Mr. FLEMING. To follow up on what the chairman was asking, you said it was stopped because of political pressures based on the scientific evidence. Is 2,4,5-T still being used in this country now?

Mr. KAHN. All but a few uses were banned by the EPA in 1977. It is still in use in Silvex on rice and a few other small restricted areas.

Mr. FLEMING. I guess my question would be, why didn't political pressure cease the use of it in this country as well as in Vietnam? Are you saying it is still being used now in forests and spraying——

Mr. KAHN. No, no. The large scale—2,4,5-T, usually mixed with 2,4-D and sometimes with other things, was used extensively in this country and was continued to be used up until 1977. At that time all use in this country, except for a few restricted ones, was stopped.

Mr. FLEMING. What restricted ones are you talking about?

Mr. KAHN. It is still used on rice crops, particularly in Louisiana, and a few rangeland applications.

Mr. FLEMING. If the danger is so great, why would there be exceptions?

Mr. KAHN. There are powerful vested interests behind it on one side, and second, the contamination in 2,4,5-T that is now used is far lower than was the case in Vietnam.

Mr. FLEMING. Is it still a danger, in your view?

Mr. KAHN. 2,4,5-T?

Mr. FLEMING. Yes. In the areas it is now being used, is it——

Mr. KAHN. No, sir, I don't think so.

Mr. FLEMING. You do not think there's a danger.

Mr. KAHN. I'm not absolutely sure of that, but I don't think so. The reason is that they have changed the production methods so that the level of contamination is so much lower now than it was, that I think the danger is largely gone under this bill. Is that correct?

Mr. FLEMING. You are proposing in here that melioidosis be included.

Mr. KAHN. Yes, nothing to do with agent orange.

Mr. FLEMING. The two cases you cited are tragic cases. I would submit to you, as a member of the staff here, we have a number of cases that come into the committee almost on a daily basis that are equally tragic that have nothing to do with agent orange.

Mr. KAHN. I believe it.

Mr. FLEMING. My question is, do you think the weight of the evidence is such on this condition as versus, let's say, soft tissue sarcomas—that it should be included in H.R. 1961?

Mr. KAHN. I'm sorry, would you——

Mr. FLEMING. You feel melioidosis should be included in the bill. Are you basing that on scientific evidence that is available now, so that we should presume disability is a result of this disease?

Mr. KAHN. Yes.

Mr. FLEMING. And you think the weight of the evidence is just as great as it is for soft tissue sarcoma, as contained in Mr. Daschle's bill?

Mr. KAHN. In the case of melioidosis, I think it is ironclad. There is no other way to get it. If you don't go to Southeast Asia, you basically don't get it.

In the case of malignant lymphoma, the evidence is not quite as strong as soft tissue sarcoma, but you have to bear in mind that a little less work has been done.

Mr. FLEMING. What kind of problems result from this disease?

Mr. KAHN. Which one?

Mr. FLEMING. Melioidosis.

Mr. KAHN. In the acute stage, it usually becomes an acute fulminating infection. Septicemia sets in and most cases are fatal. Some recover with antibiotic treatment. There is some controversy as to what is the best regimen, and that varies from case to case.

In the chronic stage of it, which can go on for years and years and never become acute, or it can erupt into acute, you can get abscesses and sores about the body in various places. In the second veteran's case whom I described, we are going to have him worked up for melioidosis because we think he is a possible candidate.

You can get abscesses of internal organs, which can lead, of course, to problems with those organs. It can appear as pneumonia. It can look like tuberculosis. It can look like malaria. It can arise manifesting as fever of unknown origin. I believe one-third of the noncombat casualties in Vietnam were fevers of unknown origin. Many of those are probably malaria attacks or misdiagnosed, but some of them probably are chronic melioidosis that was missed.

Mr. FLEMING. Then some veterans who may think they have a condition that is related to exposure to agent orange, may in fact be suffering from melioidosis.

Mr. KAHN. It may very well. The problem is that some of these peculiar things—how shall I put it.

If a man is highly susceptible to, let's say, respiratory problems, and the origin of the susceptibility is melioidosis, you cannot distinguish that from the knockdown in immune response brought about by dioxin exposure leading to the same result, respiratory problems. We have no way to distinguish those two at present. And the most frequent manifestation would be respiratory problems.

Mr. **Fleming**. But there could be other problems?

Mr. **Kahn**. Yes.

Mr. **Fleming**. Apparently a vast array of problems resulting from——

Mr. **Kahn**. Yes.

Mr. **Fleming**. Thank you.

Mr. **Applegate** [presiding]. Mr. Daschle.

Mr. **Daschle**. Mr. Kahn, isn't it true that permits are now required for the use of 2,4,5-T or 2,4-D?

Mr. **Kahn**. Yes.

Mr. **Daschle**. I was under the impression that most permits only allow point source spraying, that there is no aerial spraying; is that correct?

Mr. **Kahn**. I believe that is the case, but I haven't followed that in detail in the last year or so.

Mr. **Daschle**. Isn't it also true that there is quite a significant difference in the dilluted aspects?

Mr. **Kahn**. Well, you have two factors at work there. First, the agent orange that was used in Vietnam contained vastly more dioxin than the 2,4,5-T that is used here. Second, the concentration of defoliant applied in Vietnam was 7 to 13 times the maximum allowed concentration used in this country. It's a doublewhammy.

Mr. **Daschle**. Which, emphasizing again, requires a permit on the part of the EPA for use in any case?

Mr. **Kahn**. That is correct. And many countries around the world have banned the use of 2,4,5-T altogether.

Mr. **Daschle**. That was the proposal and, of course, there was——

Mr. **Kahn**. Great Britain, Sweden, and several more.

Mr. **Daschle**. It was overridden and now they allow a very limited use.

Thank you, Dr. Kahn, for your testimony.

Mr. W. **Wilson**. Mr. Daschle, I have a question for you, if I might. My name is Wayne Wilson for the record——

Mr. **Daschle**. Only if it's easy.

Mr. W. **Wilson**. I listened to Major Young's testimony, and he talked about the ranch handers. You know, I was an infantryman in Vietnam a couple of times and have had occasion to talk with literally thousands of ex-infantrymen. It is my understanding that large numbers of infantry troops were detailed on a regular basis, for actually periods of years, to do the dirty work in terms of preparing the chemicals and mixing them and loading them on the aircraft. The crews didn't do a lot of that.

I often wondered why those kinds of infantry troops—we do have people in New Jersey that tell us they were a part of those kinds of details—why they were never included to give a larger sample. These were people that I know didn't take a shower, that went back to the bush and stayed there for weeks. I just wondered why, if we're going to bring these Air Force fellows down to Texas, why infantry troops who did the dirty work were not included. It would have given us a larger sample.

Mr. Daschle. I think you raise a very good point. The comment made by the criticism of the National Research Council was very critical on that basis, talking about too small a sample, really, to draw any conclusions. The sample we were drawing from really was a total set of different circumstances.

It was pointed out that from time to time you may have seen somebody who was drenched in agent orange, but again, it goes to the person's ability to cleanse himself very shortly after that period of time, and even during the time of dissemination you find that—you know, if we were in an open-air biplane that would be one thing. But these pilots are encapsulated, of course, in their airplanes, and the exposure there was far less. So I think it is a legitimate criticism, as you say, and I certainly wish we could have expanded the study. I think we would have seen more legitimate results.

Mr. Kahn. Could I raise a point?

Mr. Fleming. Mr. Chairman, may I respond to that?

Mr. Daschle. Yes, Mack.

Mr. Fleming. The difference, of course, is that the Ranch Hand personnel are easily identified. It is a very simple answer to the question that you raised about the infrantrymen. The epidemiological study that is being conducted by the CDC is much more complex. You don't have any identifiable number of people. The epidemiological study by the CDC has been lost in these 2 days of hearings. The Federal Government is going to spend many millions of dollars in order to do just what you are suggesting which is the way it should be done.

But the answer to your question on the Ranch Hand study is simple—they can be identified.

Mr. W. Wilson. You know, you speak about identification. We are a State commission and we have gone out with questionnaires to some 20,000 Vietnam veterans to date. We ask them questions and we ask them to provide us with certain information. We find that we are able to identify people who flew helicopter missions, or people from Philco-Ford. Civilians even have been in touch with us. We don't seem to be having so much difficulty in identifying people who worked with chemicals other than Ranch Hand. I just wonder how much effort is being put into asking folks.

You have 16 State commissions around the country, and it seems to me that it wouldn't be all that difficult to ask us to identify people who handled or worked with these chemicals and provide the names to the VA. No one has asked us to do that, though.

Mr. Fleming. The epidemiological study that is going to be done by CDC will hopefully accomplish exactly what you are suggesting; that is, get large cohorts together, compare those who served in Vietnam with people who didn't serve in Vietnam. When we have those results we can begin to make some hard decisions as to the payment of compensation where warranted.

Mr. W. Wilson. What do I tell case A and case B?

Mr. Fleming. I don't know what you tell case A and case B. I would suggest that you remind them that Mr. Daschle 2 years ago led the efforts of the committee to provide free health care to those individuals which otherwise wouldn't have been available to them.

Congress is taking care of the health care needs of these individuals——

Mr. W. WILSON. This is a different issue. In Public Law 97-72, is that what you're saying?

Mr. FLEMING. Yes.

Mr. W. WILSON. I am sure this is not the place, but I see thousands of Vietnam veterans and have talked to thousands, and I would take exception with that. That's the real world out there.

Mr. FLEMING. The Daschle bill which became law provides that Vietnam veterans who think they are suffering from disabilities that may have resulted from exposure to agent orange——

Mr. W. WILSON. I know what the law is.

Mr. FLEMING [continuing]. Are going to receive care on a priority basis, inpatient and outpatient.

Mr. W. WILSON. Well, I would say there is a big gap between the intent of you folks up there and what is really going on out in the world. I would invite you to come to Philadelphia or New Jersey and talk with a room full of Vietnam veterans.

Mr. DASCHLE. Mr. Edgar, the chairman of the subcommittee, has hearings coming up in June for specifically that purpose. I would like to hear about it, and certainly, if the law isn't working, we want to know about it.

Mr. KAHN. Could I make a comment on case/control methods which came up for a bit of criticism a few minutes ago—that is the basis of the work that Hardell and his people have done in Sweden and several others have done around the world.

Criticism was made by the Veterans' Administration people here that case/control methods simply can't give you the answers you want, and that happens to be wrong. Case/control methods are specifically designed to give you statistical power with numbers of cases smaller than you normally employ in large-scale epidemiology, which is a complete reversal of what they said.

Moreover, in throwing a brickbat at the case/control work done on soft tissue sarcomas, Al Young then turned around and described for you the case/control methods that are being used in the Ranch Hand study, saying they were fine. If they are fine there, then they are fine anywhere else. You can't have it both ways.

Thalidamide, the drug that caused birth defects in children of women who were given it during pregnancy, was statistically significant at the 95 percent confidence level with 11 cases.

Mr. APPLEGATE. Dr. Shepard.

Dr. SHEPARD. Mr. Chairman, I just simply cannot let that last statement by Dr. Kahn, as well intentioned as it may have been, to go unchallenged.

The Ranch Hand study is not a case/control study. It is a classic cohort study.

Mr. KAHN. OK.

Mr. APPLEGATE. Thank you, Dr. Shepard.

Mr. Moon.

Mr. MOON. I have one quick question for you, Dr. Kahn, with regard to melioidosis. Do you know of a single case, where a veteran served in South Vietnam and is diagnosed as having melioidosis today, that has not been granted VA benefits? Do you know of a single case?

Mr. KAHN. No, sir.

Mr. MOON. I don't think you will find one.

Mr. KAHN. There are several published cases of Vietnam veterans who have had it—these are in the medical literature—but I don't know what disposition was made of them.

Mr. MOON. I shall not speak totally for VA policy, but I believe it to be you will find every one of those cases granted, if the facts are as you say. I just wanted to get that on the record.

Mr. KAHN. If that is the case, then——

Mr. MOON. I question the need for legislation in that area.

Mr. KAHN. If that is the case, then there is no need. I don't know of anything of that sort.

Mr. MOON. Thank you.

Mr. APPLEGATE. Thank you, Dr. Kahn. We appreciate your coming and appearing before the committee.

I just want to say thank you to all of the witnesses who appeared today and yesterday to share their views with us. They will, of course, be very important to us as we move along through the rest of the hearings on this bill. I plan to hold additional hearings on the bill later this spring, probably in June, at which time it is expected that we will have some additional and significant information to consider further. We will also have the benefit of data to be presented next month to the Subcommittee on Oversight and Special Investigations on the status of the Federal studies that are now underway.

So thank you all very much for coming, and this meeting is now adjourned.

[Whereupon, at 1:05 p.m., the subcommittee adjourned.]

# APPENDIX

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I

98TH CONGRESS
1ST SESSION

# H. R. 1961

To amend title 38, United States Code, to provide a presumption of service connection for the occurrence of certain diseases related to exposure to herbicides or other environmental hazards or conditions in veterans who served in Southeast Asia during the Vietnam era.

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## IN THE HOUSE OF REPRESENTATIVES

MARCH 8, 1983

Mr. DASCHLE (for himself, Mr. PANETTA, Mr. BONIOR of Michigan, Mr. EDGAR, Mr. LONG of Maryland, Mr. WILLIAMS of Ohio, Mr. RICHARDSON, Mr. APPLEGATE, Mr. GARCIA, Mr. KASTENMEIER, Mr. OLIN, Mr. SMITH of New Jersey, Mr. MOAKLEY, Mr. OTTINGER, Mr. WHITEHURST, Mr. BARNES, Mr. KASICH, Mr. LELAND, Mr. FEIGHAN, Mr. RATCHFORD, Mr. DUNCAN, Mr. FORD of Michigan, Mr. SMITH of Florida, Mr. FRANK, Mr. TALLON, Mr. YOUNG of Alaska, Mr. MURTHA, Mr. STARK, Mr. MORRISON of Connecticut, Mr. OWENS, Mr. SIMON, Mr. ROE, Mr. FAUNTROY, Mr. MITCHELL, Mr. SCHEUER, Mr. STUDDS, Mr. DORGAN, Mr. LAFALCE, Mr. ERDREICH, Mr. CORRADA, Mr. ECKART, Mr. FORD of Tennessee, Mr. TRAXLER, Mr. SPRATT, Mr. WILLIAMS of Montana, Mr. OBERSTAR, Ms. MIKULSKI, Mr. PERKINS, Mr. LOWRY of Washington, Mr. MCKINNEY, Mr. HERTEL of Michigan, Mr. WIRTH, Mrs. SCHNEIDER, Mr. LANTOS, Mr. MARTINEZ, Mr. BERMAN, Mr. SHANNON, Mr. KILDEE, Mr. D'AMOURS, Mrs. COLLINS, Mr. FOGLIETTA, Mr. LEVINE of California, Mr. WEISS, Mr. HARRISON, Mr. FAZIO, Mr. MINETA, Mr. STOKES, Mr. DWYER of New Jersey, Mr. MURPHY, Mr. WEAVER, Mr. MCHUGH, Mr. HOWARD, Mr. DURBIN, Mr. MARKEY, Mr. BATES, Mr. SEIBERLING, Mr. VENTO, Mr. BORSKI, Mr. DONNELLY, Mr. SUNIA, Mr. MRAZEK, Mr. ADDABBO, Mr. RODINO, Mrs. KENNELLY, Mr. BEREUTER, Mr. DELLUMS, Mrs. BOXER, Mr. EDWARDS of California, Mr. JEFFORDS, Mr. BROWN of California, Mr. SHARP, Mr. PENNY, Mr. FISH, Mr. ASPIN, Mr. KEMP, Mr. FLORIO, Ms. OAKAR, Mr. GORE, Mr. HARKIN, Mr. OXLEY, Mr. DE LUGO, Mr. MCCLOSKEY, Mr. GUNDERSON, Mr. GILMAN, Mr. KOGOVSEK, Mr. TORRICELLI, Mr. HORTON, Mr. DIXON, Mr. EVANS of Illinois, Mr. MILLER of California, Mr. WOLPE, Mr. GLICKMAN, and Mr. SLATTERY) introduced the following bill; which was referred to the Committee on Veterans' Affairs

# A BILL

To amend title 38, United States Code, to provide a presumption of service connection for the occurrence of certain diseases related to exposure to herbicides or other environmental hazards or conditions in veterans who served in Southeast Asia during the Vietnam era.

*Be it enacted by the Senate and House of Representatives of the United States of America in Congress assembled,* That this Act may be cited as the "Vietnam Veterans Agent Orange Relief Act".

SEC. 2. The Congress finds that—

(1) certain adverse health effects occurring among persons who served in the Armed Forces in Southeast Asia during the Vietnam era, and certain birth defects occurring among the children of such persons, may be the result of the exposure of such persons during such service to phenoxy herbicides (including the herbicide known as Agent Orange) and the class of chemicals known as the dioxins produced during the manufacture of such herbicides or to other factors involved in such service including exposure to other herbicides, chemicals, medications, or environmental hazards or conditions; and

(2) a comprehensive review and scientific analysis of the literature covering studies relating to whether there may be long-term adverse health effects in

humans from exposure to any of the class of chemicals known as the dioxins produced during the manufacture of the various phenoxy herbicides (including the herbicide known as Agent Orange), as required by section 307(a)(1)(B) of Public Law 96–151, has been completed and submitted to the Veterans' Administration.

SEC. 3. Section 312 of title 38, United States Code, is amended by adding at the end the following new subsection:

"(d)(1) For the purposes of section 310 of this title and subject to the provisions of section 313 of this title, in the case of a veteran who served in Southeast Asia during the Vietnam era and who after such service suffers from a disease described in paragraph (2)(A) of this subsection, such disease shall be considered to have been incurred in or aggravated by such service, notwithstanding that there is no record of evidence of such disease during the period of service.

"(2)(A) The diseases referred to in paragraph (1) of this subsection are the following:

"(i) Soft-tissue sarcomas.

"(ii) Porphyria cutanea tarda.

"(iii) Active and residual chloracne and chloracneform lesions.

"(iv) A disease listed in a regulation prescribed by the Administrator under subparagraph (B) of this paragraph.

"(B) The Administrator may determine, and prescribe by regulation, diseases (in addition to those listed in subparagraph (A) of this paragraph) that medical research has shown may be due to exposure to herbicides, chemicals, medications, or environmental hazards or conditions. The Administrator shall include in such regulations a specification of the standards used by the Administrator in making such determination.

"(3) Paragraph (1) of this subsection shall terminate on the first day of the first month beginning after the end of the one-year period beginning on the date the Administrator submits to the appropriate committees of Congress the first report required by section 307(b)(2) of the Veterans Health Programs Extension and Improvement Act of 1979 (Public Law 96-151; 93 Stat. 1098).".

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Office of the
Administrator
of Veterans Affairs

Washington DC 20420

**Veterans
Administration**

APRIL 25 1983

Honorable G. V. (Sonny) Montgomery
Chairman, Committee on
Veterans' Affairs
House of Representatives
Washington, D.C. 20515

Dear Mr. Chairman:

I am pleased to present the views of the Veterans Administration on H.R. 1961, 98th Congress, the proposed "Vietnam Veterans Agent Orange Relief Act." I share with you and other members of Congress the desire for a meaningful Federal response to the fears of veterans who served in Vietnam that their exposure to Agent Orange may have long-term adverse effects on their health. However, we consider the approach taken in H.R. 1961 inadvisable given the present state of scientific knowledge.

The controversies arising from the Government's use of Agent Orange in Vietnam are not yet resolved. Before turning to the Veterans Administration's observations concerning the several issues raised by H.R. 1961, I would like to emphasize that the potential cost of paying compensation based on any Agent-Orange-caused disabilities played no part in our deliberations on this measure. The Federal Government, since its beginning, has fulfilled its sacred obligation to veterans disabled in the line of duty and will continue to do so.

The devastating wars of this century, and the need to maintain peacetime forces in order to assure the defense of our Nation, have been accompanied by legislative and programmatic developments intended to assure that no veteran's reasonable claim to compensation is denied. This is true whether the disability results from a combat wound, service-incurred disease, in-service accident, psychological trauma resulting from combat or other conditions of military service, or exposure to a substance known or later discovered to have adverse health effects.

We are immensely proud of our Agency's record of achievement. It can safely be maintained that our compensation program is the finest in the world, both in terms of the number of veterans we serve and in the amount of benefits paid. Moreover, the American people--who fund this program with their taxes--have given it overwhelming support, as has the Congress of the United States.

The preservation and integrity of the compensation program are among the highest priorities of the Veterans Administration.

There are certainly many veterans suffering from illnesses they ascribe to exposure to that herbicide, especially its containant dioxin. Although scientific evidence is lacking, there are persons in the medical and scientific communities who contend that exposure may lead to a host of disorders that appear long after the exposure has ceased. There are also organizations and individuals who believe very sincerely that the Veterans Administration has not responded adequately to the issues involved.

As guardians of the public trust, Congress and the Administration share, I believe, a commonality of aims respecting these issues. The compensation program must be attuned to justifiable conclusions about the connection between Agent Orange exposure and disorders possibly arising from that exposure. At the same time we must do our best to avoid taking steps that have the potential for undermining the program's credibility and legitimacy because of inconclusive scientific evidence. I know that you and other Members will give careful and thorough consideration to the bill, keeping in mind the commonality of aims to which I have previously alluded.

H.R. 1961 is intended to assist veterans who served in Southeast Asia during the Vietnam era establish entitlement to service-connected disability compensation if they are currently suffering from one of the disorders specified in the bill. It would do this by amending section 312 of title 38, United States Code, in order to provide for a special presumption of service connection applicable only to these veterans.

The bill is based on the premise that each of the specified disorders, no matter how long after military service symptoms appear, can be attributed to exposure to a phenoxy herbicide in service. During the period 1962 to 1971, phenoxy herbicides, including Agent Orange, were used in Vietnam. As I have noted, H.R. 1961 is an effort to respond to the widespread concern that exposure to Agent Orange, especially its contaminant dioxin, may have long-term adverse effects on veterans' health.

Authority to award compensation on the basis of the presumption provided for in the bill would terminate one year after submission to Congress of the comprehensive epidemiological study mandated by Pub. L. No. 96-151. This "sunset" provision is analogous to the sunset provision applicable to VA health care for certain disorders possibly associated with phenoxy herbicide exposure, authorized by Pub. L. No. 97-72. Both sunset provisions recognize the current uncertainties as to the long-term effects of exposure.

The Agent Orange controversy, as it relates to individual veterans' compensation claims, involves two basic questions: (1) whether the veteran was exposed, and (2) whether the veteran's disability results from the exposure. H.R. 1961, it should be noted, does not require any evidence of exposure; it would afford the presumption to any veteran who served in Southeast Asia during the Vietnam era (1964-1975). We have previously made public our decision to resolve the issue of exposure in a manner favorable to veterans; unless there is affirmative evidence to the contrary, we are prepared to presume exposure if a veteran served in Vietnam during the relevant period. This policy, prompted by the lack of a definitive method for identifying exposed individuals, is consistent with our longstanding policy of giving veterans the benefit of the doubt.

There may be, however, some cases in which affirmative evidence refutes even the possibility of exposure, and, therefore, our policy is necessarily qualified. The lack of any similar qualification in H.R. 1961, in our view, is unjustifiable. We observe also that affording the presumption to veterans who served in Southeast Asia--a far broader region than Vietnam, embracing areas where no phenoxy herbicides were used--inappropriately expands the category of veterans intended to be benefited.

Our principal concerns, however, relate to the concept of an open-ended presumption that would be established by the bill and to the conclusions it embodies as to the specific disorders chloracne, porphyria cutanea tarda (PCT), and the several malignancies grouped as soft-tissue sarcoma. (The bill would also authorize presumptive service connection for additional disorders, provided for by regulation, that "medical research" has shown "may be" attributable to chemical exposure or environmental hazards or conditions. This requirement is vague--rules creating such presumptions should be based only on well-accepted, scientifically valid findings--and also unnecessary in view of the Administrator's current authority to issue regulations.)

The post-service presumption periods provided for in section 312(a) of title 38 are appropriate for chronic diseases whose inception in service may not be recorded because the development of pathology is gradual and insidious. They are justifiable when reasonably supported by medical knowledge as to the pathological courses of the particular diseases.

Congress has wisely set time limits on these presumptive provisions; unless symptoms of the disease appear within a specified period of time after service, the presumption is not available. The section 312(a) presumption, together with the time limits, assures that no veteran's reasonable claim is overlooked but also does not dictate grants of service connection when there is no evidence of service incurrence and it is not reasonable to infer service origin.

Reputable studies have concluded that dioxin exposure may result, within a relatively short period, in chloracne. PCT resulting from exposure also appears within a few weeks. On the other hand, no studies have shown that exposure results in the initial appearance of these disorders after lengthy delays. Our current authorities are adequate, without the need of a presumption, to award service connection and compensation, if appropriate, in cases of chloracne or PCT appearing within expected time limits after the exposure. Requiring us to award service connection for initial occurrence of these disorders long after the exposure incidents is, we believe, unjustifiable in the absence of any evidence indicating they are latent effects of exposure.

As I have noted, individuals in whom these relatively rare disorders appear begin to suffer symptoms soon after exposure, ordinarily within days or weeks. Chloracne is a skin disorder caused by exposure to certain chlorine-containing chemicals, including dioxin. In its more serious manifestations, it causes discomfort and disfigurement. Most cases clear up within a year or two after the exposure ceases, but in a few, the disorder persists. The Veterans Administration acknowledges that chloracne can result from exposure to Agent Orange during service in Vietnam and has established procedures to assure careful and liberal consideration of all claims based on this disorder.

Since 1978, we have awarded service connection in 1,225 skin-disorder cases involving veterans who served in Vietnam. We have scrutinized more than 3,000 claims for service-connected benefits to determine whether there are indications of chloracne. Those cases in which it was believed this diagnosis was at least possible were further reviewed by a VA dermatologist, and 13 have been examined in person by dermatologists at prestigious private clinics.

Although all of these cases involve skin disorders of various types and all involve veterans who served in Vietnam, only one case of possible chloracne has been identified. We will, of course, continue our investigations of this issue.

H.R. 1961 would also extend presumption of service connection for "chloracneform lesions." This is a term not found in medical or scientific literature, but can be taken to mean "lesions resembling chloracne." As certain common skin disorders may resemble chloracne, this term is overly broad and would, we believe, cause unnecessary confusion.

PCT, an uncommon liver disorder, can be triggered by exposure to various chemicals including alcohol. There is no evidence that PCT is a latent effect of exposure. Each attack ordinarily subsides in about a year after contact with the chemical ceases, but prolonged exposure, as in chronic alcoholism, may cause permanent damage to the liver. An attack of PCT induced by Agent Orange or exposure to any other chemical during service in Vietnam years ago would not be expected to impair a veteran's health today. As is the case with chloracne, we regard our current authorities as fully adequate to assure proper consideration of PCT claims based on exposures during military service. As a technical matter, the proper application of section 313 of title 38, United States Code, making section 312 presumptions rebuttable if there is evidence of an intercurrent cause, would reduce the likelihood of awards of service connection based on the PCT presumption, if enacted.

The issue as to whether the malignancies grouped as "soft-tissue sarcomas" result from phenoxy herbicide exposure presents a problem of far greater complexity. There is considerable uncertainty in the scientific community on this issue. Advocates of the belief that exposure "causes" soft-tissue sarcoma generally cite studies involving cancer victims believed to have been exposed to phenoxy herbicides whose first symptoms appeared long after the exposure. Because it is well established that exposure to radiation and other agents like asbestos and benzene may result in the latent development of malignancy, these advocates reason by analogy that phenoxy herbicide exposure "causes" soft-tissue sarcoma. The vital question is, therefore, the weight that should be given to the studies they cite.

"Soft tissue sarcomas" are a group of malignant tumors, or cancers. Any sarcoma arises in a body cell that does not cover a body surface, form glandular tissue, or line certain body cavities. "Soft tissue" excludes sarcomas in "hard tissues" such as bone or cartilage. Hence, soft tissue sarcomas arise from such body tissues as muscles, tendons, blood vessels, fat, and connective tissues.

Certain cancers share some characteristics of soft-tissue sarcomas but are not placed in that group. These include most brain tumors and the so-called blood cancers, chiefly the leukemias. Some authorities include tumors of the lymph nodes--the lymphomas--with the soft-tissue sarcomas. The World Health Organization "International Classification of Tumors, No. 3, Histological Typing of Soft Tissue Tumors," however, excludes lymphomas and appears to be adequate for purposes of defining the malignancies in this category.

There is no evidence that all soft-tissue sarcomas have a common etiology or cause. These malignancies differ from one another as to how rapidly they grow and spread, how they are treated, and the results that treatment achieves. However, all are considered lethal if not successfully treated.

These malignancies are rare. According to the National Cancer Institute, they comprise 2.76 percent of all cancer cases in men aged 25 to 29 and 0.58 percent of all cancer cases in men aged 55 to 59; the percentage declines because other types of cancers become increasingly common with age. Lymphomas, sometimes included with soft-tissue sarcomas, contribute another 5.21 percent at ages 25 to 29 and 2.40 percent at ages 55 to 59.

Although there is no evidence establishing a common cause for these sarcomas, some malignancies in the group are known to be associated with exposure to environmental hazards. For example, malignant mesothelioma is known to be caused by asbestos exposure, and angiosarcoma of the liver by exposure to vinyl chloride.

Because these malignancies are rare, it is difficult to devise adequate techniques to investigate their causes. A series of studies in Sweden using the "case/control" method grouped the soft-tissue sarcomas together in order to investigate whether Swedish foresters and farmers exposed to herbicides and a chemical known as chlorophenol in their work, suffered latent malignancies of this type. These studies have been carefully reviewed by Richard D. Remington, Dean of the School of Public Health, University of Michigan, at the request of the Office of Technology Assessment and determined to have been carefully conducted and well reported with results that suggest a relationship between herbicide exposure and soft-tissue sarcomas. Significantly, Dr. Remington pointed out the limitations of the case/control methodology and found the Swedish studies inadequate to permit definite conclusions.

Investigations in the United States based on studies of industrial workers have also suggested a phenoxy-compound connection with soft-tissue sarcomas. In addition, an East German investigation of malignant neoplasms among pesticide sprayers and agricultural technicians tends to support the Swedish studies by finding a single case of soft-tissue "malignancy," which probably was a soft-tissue sarcoma.

Other studies, in Finland, New Zealand, Great Britain, the Netherlands, and Italy have not confirmed the Swedish studies. In addition, a separate investigation of Swedish forestry workers casts some doubt on the Swedish studies.

We do not disagree with Dr. Remington's conclusions as to the credibility and limitations of the Swedish studies. They lay a predicate for further investigation and do not rule out the possibility of a causal link. They do not, however, provide a reasonable basis upon which to favorably decide VA compensation claims.

We recognize the importance of careful scientific analysis in matters of this kind, and have appended to this report detailed background papers concerning these diseases.

The comprehensive epidemiological study mandated by Pub. L. No. 96-151, together with other ongoing studies including some devoted specifically to the soft-tissue sarcoma issue, may resolve many of the controversial questions raised by the use of Agent Orange in Vietnam. As I stated at the outset of this report, we must work toward the dual objectives of fair compensation for any Agent-Orange-caused disabilities and avoidance of steps that would compromise the integrity of the program. At this point, there is no evidence that either chloracne or PCT is a delayed effect of exposure, and we believe the provisions of H.R. 1961 respecting these disorders are not justified. We do not believe it has been satisfactorily demonstrated that exposure can cause soft-tissue sarcoma.

Accordingly, we oppose the enactment of H.R. 1961. In view of the current state of scientific findings, enactment would compromise the integrity of the compensation program and engender unfounded fears among Vietnam veterans that lethal illnesses may yet befall them as a result of having answered duty's call. Our biding moral obligation to veterans who have given so much demands that we act responsibly in all matters affecting the compensation program.

If the soft-tissue sarcoma presumption in H.R. 1961 were to be enacted, we estimate compensation benefit costs in fiscal year 1984 ranging from $2 million to $11 million, with the range for DIC benefits $3.5 million to $18.7 million. Benefit costs for future fiscal years would be comparable. A range of estimates is necessary because of uncertainty as to which malignancies are to be covered. Administrative costs would be sizeable in the first fiscal year and are anticipated to be $6.2 million, but would level off during subsequent fiscal years to less than $600,000 in fiscal year 1988.

Costs relating to chloracne are estimated as insignificant. We can only speculate with regard to costs resulting from the inclusion of "chloracneform lesions."

As PCT is a relatively uncommon disorder, we would not anticipate benefit costs exceeding $1 million in any fiscal year from the PCT presumption.

Advice has been received from the Office of Management and Budget that there is no objection to the presentation of this report from the standpoint of the Administration's program.

Sincerely,

Harry N. Walters

HARRY N. WALTERS
Administrator

Enclosure

DEPARTMENT OF HEALTH & HUMAN SERVICES

Office of the Secretary

Washington, D.C. 20201

February 24, 1983

MAR 4 1983

MEMORANDUM TO: All State Veterans Departments/
State Agent Orange Commissioners/
Veterans Service Organizations and
Coordinators, etc.

SUBJECT : Update on Agent Orange Fact Sheet

The attached Fact Sheet has been updated and revised, since the July 7, 1982 release and was compiled with the assistance of several members of the Science Panel of the Cabinet Council Agent Orange Working Group. It provides a listing of Federal Research into two broad categories:

a) Human Studies; and,
b) Other Studies.

This Fact Sheet may be helpful to you and your organization.

Please circulate to any group you represent.

Dr. Peter E.M. Beach
Director of Veterans Affairs/HHS
Staff Director
Agent Orange Working Group

Attachment

CABINET COUNCIL ON HUMAN RESOURCES
AGENT ORANGE WORKING GROUP

FEDERALLY SPONSORED HUMAN STUDIES RELATED TO AGENT ORANGE

| AGENCY | TYPE OF STUDY | | | | | STATUS | | |
|---|---|---|---|---|---|---|---|---|
| STUDY TITLE | Mortality | Morbidity | Cancer | Reproduction | Analytical | Completed | Ongoing | Estimated Completion Date |
| DEPARTMENT OF HEALTH AND HUMAN SERVICES | | | | | | | | |
| NIEHS Investigation of Leukemia Cluster in Madison County, Kentucky Allegedly Associated with Pentachlorophenol Treated Ammunition Boxes | | | X | | | X | | Publications in Press |
| CDC Birth Defects and Military Service in Vietnam Study | | | | X | | | X | Late 1983 |
| NIOSH Dioxin Registry | X | | X | | | | X | Indefinite |
| NIOSH Establishment and Maintenance of an International Register of Persons Exposed to Phenoxy Acid Herbicides and Contaminants | X | | X | | | | X | Indefinite |
| NIOSH Soft Tissue Sarcoma Investigation | | | X | | | | X | Indefinite |
| NCI Case Control Study of Lymphoma and Soft Tissue Sarcoma | | | X | | | | X | Indefinite |
| NCI Study of Mortality Among Pesticide Applicators from Florida | | | | | | X | | Publications in Press |

FEDERALLY SPONSORED HUMAN STUDIES RELATED TO AGENT ORANGE

| AGENCY | TYPE OF STUDY | | | | | STATUS | | |
|---|---|---|---|---|---|---|---|---|
| STUDY TITLE | Mortality | Morbidity | Cancer | Repro-duction | Analytical | Completed | Ongoing | Estimated Completion Date |
| DEPARTMENT OF HEALTH AND HUMAN SERVICES cont'd | | | | | | | | |
| *CDC Epidemiologic Study of Ground Troops Exposed to Agent Orange during the Vietnam Conflict | X | X | X | X | | | X | 1987 |
| VETERANS ADMINISTRATION | | | | | | | | |
| Vietnam Veteran Mortality Studies | X | | | | | | X | Late 1984 |
| Vietnam Veteran Identical Twin Studies | | X | | X | | | Proto-col | Initial 1984 |
| Survey of Patient Treatment File for Vietnam Veteran In-Patient Care | | X | X | | | | X | Initial 1983 Survey |
| Agent Orange Registry Examinations | | X | X | | | | X | Indefinite |
| TCDD in Body Fat of Vietnam Veterans and Other Men | | X | | X | X | | | Publication in Preparation |
| Retrospective Study of Dioxins and Furans in Adipose Tissue of Vietnam-Era Veterans | | | | | X | | X | 1985 |

*Mandated to the VA by P.L. 96-151 Sec. 307. Transferred from VA to CDC under Interagency Agreement January 14, 1983.

FEDERALLY SPONSORED HUMAN STUDIES RELATED TO AGENT ORANGE

| AGENCY | TYPE OF STUDY | | | | | STATUS | | |
|---|---|---|---|---|---|---|---|---|
| STUDY TITLE | Mortality | Morbidity | Cancer | Reproduction | Analytical | Completed | Ongoing | Estimated Completion Date |
| DEPARTMENT OF DEFENSE | | | | | | | | |
| Epidemiologic Investigation of Health Effects in Air Force Personnel Following Exposure to Herbicide Orange (Air Force Health Study) | X | X | X | X | | | X | Baseline 1983<br>Complete 1999 |
| Armed Forces Institute of Pathology Agent Orange Registry of Vietnam Veteran Biopsy Tissues | | | X | | | | X | Indefinite |
| ENVIRONMENTAL PROTECTION AGENCY | | | | | | | | |
| Report of Assessment of a Field Investigation of Six-Year Spontaneous Abortion Rates in Three Oregon Areas of Relation to Forest 2,4,5-T Spray Practices | | | | X | | X (Published) | | |
| National Pesticide Monitoring Project of Human Adipose Tissue | | | | | X | | X | Indefinite (Annual Reports) |

FEDERALLY SPONSORED HUMAN STUDIES RELATED TO AGENT ORANGE

| AGENCY | TYPE OF STUDY | | | | | STATUS | | |
|---|---|---|---|---|---|---|---|---|
| STUDY TITLE | Mortality | Morbidity | Cancer | Repro-duction | Analytical | Completed | Ongoing | Estimated Completion Date |
| DEPARTMENT OF AGRICULTURE | | | | | | | | |
| A Case Control Study of the Relationship Between Exposure to 2,4-D and Spontaneous Abortions in Humans | | | | X | | X | | |
| Exposure Measurements of Mixers, Loaders and Applicators of 2,4-D on Wheat | | | | | X | | X | 1982 |
| Exposure of Forest Workers to Ground Applications of 2,4-D | | | | | X | | X | 1983 |

FEDERALLY SPONSORED LABORATORY STUDIES AND LITERATURE SURVEYS RELATED TO AGENT ORANGE

| AGENCY | TYPE OF STUDY | | | | STATUS | | |
|---|---|---|---|---|---|---|---|
| STUDY EFFORT | Animal | Environmental | Analytical | Literature | Completed | Ongoing | Estimated Completion Date |
| DEPARTMENT OF HEALTH AND HUMAN SERVICES | | | | | | | |
| Bioassay of Octachlorodibenzo-p-dioxin | X | | | | | X | |
| Carcinogenesis Bioassay of 2,3,7,8-Tetrachlorodibenzo-p-dioxin in Swiss Webster Mice | X | | | | | X | |
| Carcinogenesis Bioassay of 2,3,7,8-Tetrachlorodibenzo-p-dioxin in Osborne-Mendel Rats and B6C3F1 Mice | X | | | | X | | |
| Bioassay of a Mixture of 1,2,3,6,7,8- and a Mixture of 1,2,3,6,7,8-Hexachlorodibenzo-p-dioxins for Possible Carcinogenicity | X | | | | | X | |
| Comparative species Evaluation of Chemical Disposition and Metabolism of 2,3,7,8-Tetrachlorodibenzofuran (TCDF) in Rat, Monkey, Guinea Pig and Two Strains of Mice | X | | | | X | | |
| Neurotoxicity of 2,4,-D in Rodents | X | | | | X | | |

## FEDERALLY SPONSORED LABORATORY STUDIES AND LITERATURE SURVEYS RELATED TO AGENT ORANGE

| AGENCY | TYPE OF STUDY | | | | STATUS | | |
|---|---|---|---|---|---|---|---|
| STUDY EFFORT | Animal | Environmental | Analytical | Literature | Completed | Ongoing | Estimated Completion Date |
| DEPARTMENT OF HEALTH AND HUMAN SERVICES cont'd | | | | | | | |
| Studies of the Chemical Disposition and Metabolism of Octachlorodibenzodioxin (OCDD) | X | | | | | X | |
| Effects of Agent Orange Components on Male Fertility and Reproduction | | X | | | X | | |
| Mutagenicity Studies of TCDD, 2,4-D; 2,4,5-T and Esters of 2,4-D and 2,4,5-T | | X | | | | X | |
| Implications of Low Level Exposure of Dioxins | | X | | | | X | |
| Mechanisms of Toxicity of the Chlorinated- p-dioxins | | X | | | | X | |
| Research Toward Understanding the Molecular Level Mechanisms of Toxicity of TCDD and Related Compounds | X | | | | | X | |
| Synthesis of Selected Tetrachlorodibenzo-p-dioxins and Related compounds as Analytical Standards | | | X | | | X | |

FEDERALLY SPONSORED LABORATORY STUDIES AND LITERATURE SURVEYS RELATED TO AGENT ORANGE

| AGENCY | TYPE OF STUDY | | | | STATUS | | |
|---|---|---|---|---|---|---|---|
| STUDY EFFORT | Animal | Environmental | Analytical | Literature | Completed | Ongoing | Estimated Completion Date |
| DEPARTMENT OF HEALTH AND HUMAN SERVICES cont'd | | | | | | | |
| Matrix Effect and Sub Parts-per-billion Quantitative Analysis of TCDD by Mass Spectrometry - With Special Reference to Milk | X | | X | | | X | |
| Toxic Actions of Tetra-chloroazobenzene Dioxins | X | | | | | X | |
| Xenobiotic Induction of Pleiotropic Responses in Liver | X | | | | | X | |
| Molecular, Biochemical Actions of Chlorinated-p-dioxins | X | | | | | X | |
| Mechanism(s) for Toxicity of Chlorinated Dibenzo-dioxins | X | | | | | X | |

FEDERALLY SPONSORED LABORATORY STUDIES AND LITERATURE SURVEYS RELATED TO AGENT ORANGE

| AGENCY | TYPE OF STUDY | | | | STATUS | | |
|---|---|---|---|---|---|---|---|
| STUDY EFFORT | Animal | Environmental | Analytical | Literature | Completed | Ongoing | Estimated Completion Date |
| ENVIRONMENTAL PROTECTION AGENCY | | | | | | | |
| Evaluation of Large Scale Combustion Sources | | X | X | | | X | |
| Evaluation of Municipal Waste Combustors | | X | | | | X | |
| Bacterial Decomposition of TCDD | | X | | | | X | |
| Investigation of Bioavailability to Fresh Water Fish of TCDDs in Fly Ash | X | X | | | | X | |
| Analysis of Environmental Samples for PCDDs and PCDFs | | X | X | | | X | |
| DEPARTMENT OF AGRICULTURE | | | | | | | |
| Survey of Phenoxy Herbicide Use by Agricultural Commodity | | | | X | | X | |
| Survey of Phenoxy Herbicide Literature | | | | X | | X | Annaul Bibliographies Published |

FEDERALLY SPONSORED LABORATORY STUDIES AND LITERATURE SURVEYS RELATED TO AGENT ORANGE

| AGENCY | TYPE OF STUDY | | | | STATUS | | |
|---|---|---|---|---|---|---|---|
| STUDY EFFORT | Animal | Environmental | Analytical | Literature | Completed | Ongoing | Estimated Completion Date |
| DEPARTMENT OF AGRICULTURE cont'd | | | | | | | |
| Photolysis of 2,4,5-T | | | X | | | X | |
| Biological and Economic Assessment of 2,4,5-T and Silvex | | | | X | | X | |
| TCDD Residue Monitoring in Deer | X | X | | | X | | Report in Preparation |
| DEPARTMENT OF DEFENSE | | | | | | | |
| Environmental Chemistry of Herbicide orange and TCDD | | X | X | | | X | Indefinite |
| VETERANS ADMINISTRATION | | | | | | | |
| Review of Literature on Herbicides, Including Phenoxy Herbicides and Associated Dioxins | | | | X | Published 1981 | | Annual Update Approved |
| Urinary 6-Hydroxy Cortisol: Physiological and Pharmacologic Studies (Including Agent Orange) | X | | | | | X | 1982 |
| Effect of TCDD on Lipid Metabolism | X | | | | | X | 1983 |

FEDERALLY SPONSORED LABORATORY STUDIES AND LITERATURE SURVEYS RELATED TO AGENT ORANGE

| AGENCY | TYPE OF STUDY | | | | STATUS | | |
|---|---|---|---|---|---|---|---|
| STUDY EFFORT | Animal | Environmental | Analytical | Literature | Completed | Ongoing | Estimated Completion Date |
| VETERANS ADMINISTRATION cont'd | | | | | | | |
| Mechanisms of Dioxin Induced Toxicity Using the Chloracne Model - Phase I | X | | | | X | | Publication in Press |
| Behavioral Toxicity of An Agent Orange Component 2,4-D | X | | | | | X | 1984 |
| Effects of 2,3,7,8-Tetrachlorodibenzodioxin on Hepatobiliary Function in Animals | X | | | | | X | 1985 |
| Mechanism of TCDD Absorption and Toxicity on Lipid and Lipoprotein Metabolism | X | | | | | X | 1985 |
| Metabolism of the Herbicides Present in Agent Orange and Agent White | X | | | | | X | 1985 |
| TCDD Exposed Rhesus Monkeys: Effects on Behavior and Stress Hormones | X | | | | | X | 1985 |

FEDERALLY SPONSORED LABORATORY STUDIES AND LITERATURE SURVEYS RELATED TO AGENT ORANGE

| AGENCY | TYPE OF STUDY | | | | STATUS | | |
|---|---|---|---|---|---|---|---|
| STUDY EFFORT | Animal | Environmental | Analytical | Literature | Completed | Ongoing | Estimated Completion Date |
| VETERANS ADMINISTRATION cont'd | | | | | | | |
| Neuromuscular Toxicity of Agent Orange | X | | | | | X | 1985 |
| Mechanisms of Dioxin Induced Toxicity Using the Chloracne Model - Phase II | X | | | | | X | 1985 |
| Effects of Low Dose TCDD on Mammalian Chromosomes and Liver Cells | X | | | | | X | 1986 |
| Mechanism of Porphyria Caused by TCDD and Related Chemicals | X | | | | | X | 1986 |
| Effects of Agent Orange on Sleep | X | | | | | X | 1986 |

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# STATEMENT OF THE
# HONORABLE DAVID E. BONIOR
# BEFORE THE
# SUBCOMMITTEE ON COMPENSATION, PENSION, AND INSURANCE
# OF THE
# COMMITTEE ON VETERANS' AFFAIRS

APRIL 26, 1983

MR. CHAIRMAN, I AM HONORED TO HAVE THE OPPORTUNITY TO APPEAR AGAIN BEFORE THIS COMMITTEE AND ADDRESS THE AGENT ORANGE ISSUE. THIS COMMITTEE HAS PLAYED A VITAL ROLE IN AGENT ORANGE POLICY.

ITS LEADERSHIP HAS HELPED FORCE AN AGGRESSIVE RESEARCH AGENDA ON AN ALL TOO OFTEN HESITANT VETERANS ADMINISTRATION (VA). TWO YEARS AGO, IN A LANDMARK EFFORT, THIS COMMITTEE PROVIDED INTERIM ELIGIBILITY FOR HEALTH CARE FOR VIETNAM VETERANS WITH AGENT ORANGE RELATED PROBLEMS.

ENACTMENT OF H.R. 1961 IS CONSISTENT WITH THIS TRADITION OF LEADERSHIP. I COMMEND THE PRESENT CHAIRMAN OF THE VIETNAM VETERANS IN CONGRESS (VVIC), MR. DASCHLE, THE CHAIRMAN OF THIS SUBCOMMITTEE, MR. APPLEGATE, AND THE CHAIRMAN OF THE SUBCOMMITTEE ON HOSPITALS AND HEALTH CARE, MR. EDGAR, FOR THEIR ROLE IN THE DEVELOPMENT OF THIS LEGISLATION, NOW COSPONSORED BY MORE THAN 160 MEMBERS OF THE HOUSE.

## THE LONG RAOD TO H.R. 1961

MR. CHAIRMAN, OVER THREE (3) YEARS AGO, TWO INDEPENDENT SWEDISH EPIDEMIOLOGICAL STUDIES RELATED EXPOSURE TO 2,4,5-T, A MAIN INGREDIANT IN AGENT ORANGE, TO SOFT TISSUE CANCERS. DESPITE THE FACT THAT THE STUDIES WERE IN THE PUBLIC DOMAIN, INDEED, WERE PART OF THE ENVIRONMENTAL PROTECTION AGENCY (EPA) CASE AGAINST 2,4,5-T, THE VA DID NOT EVEN KNOW OF THEIR EXISTENCE.

YET THE VA WAS NOT MOVED BY THE EVIDENCE NOR EMBARRASSED BY THEIR IGNORANCE. IN AN ALMOST APPARENT RUSH TO DISMISS THE FACTS, A SCANT TWELVE (12) DAYS AFTER RECEIVING THE STUDIES, THE VA ADMINISTRATOR CONCLUDED:

> ... FOLLOWING PRELIMINARY ANALYSIS MY SCIENTIFIC ADVISERS HAVE INFORMED ME THAT THEY DO NOT THINK THE PAPERS MAKE A MAJOR CONTRIBUTION TO ANSWERING THE PROBLEM THAT IS OF CONCERN TO US. I HAVE BEEN ADVISED THAT THESE STUDIES APPEAR TO BE BASED ON A RANDOM DISTRIBUTION.

NOT EVERYONE AGREED WITH THE VA ASSESSMENT. SOME THREE MONTHS LATER, THE DISTINGUISHED OFFICE OF TECHNOLOGY ASSESSMENT (OTA) AND THE INTERAGENCY WORK GROUP ON PHENOXY HERBICIDES (WORK GROUP) COMPLETED THEIR OWN REVIEW OF THE TWO STUDIES. IN THE WORDS OF THE OTA EXAMINER:

> (THE STUDIES ARE) AMONG THE MOST CAREFULLY CONDUCTED INVESTIGATIONS OF THEIR TYPE THAT I HAVE EVER SEEN. _IN TOTO_, THE SWEDISH WORK IS CREDIBLE IF NOT CONCLUSIVE.

THE EVIDENCE HAS CONTINUED TO MOUNT. OVER ONE YEAR AGO, AN INDEPENDENT REVIEW OF FOUR ADDITIONAL GROUPS OF EXPOSED AMERICAN WORKERS FOUND THE SAME CORRELATION. NEARLY ONE YEAR AGO, IN THE DISTINGUISHED NEW ENGLAND JOURNAL OF MEDICINE, TWO DOCTORS FROM EMORY UNIVERSITY REPORTED ON THREE VIETNAM VETERANS WITH SOFT TISSUE SARCOMAS.

YET EVEN TODAY, THE VA HAS NOT YET BEEN MOVED TO PROVIDE COMPENSATION. VIETNAM VETERANS HAVE WAITED LONG ENOUGH FOR THE UNMOVABLE VA TO BE TOUCHED BY SHAME, IF NOT COMPASSION. H.R. 1961

IS A RESPONSIBLE ATTEMPT TO GIVE VIETNAM VETERANS A CLEAR SIGN THAT CONGRESS, AT LEAST, IS PREPARED TO ACT.

H.R. 1961 IS NOT THE HURRIED ANSWER TO A NEW PROBLEM. IT DOES NOT OPEN THE FLOOD GATE TO FUTURE CLAIMS NOR DOES IT REJECT SCIENCE IN THE NAME OF COMPASSION. RATHER, H.R. 1961 IS THE CONSIDERED PRODUCT OF LONG YEARS OF FRUSTRATION.

AT A YEARLY COST OF JUST $4 MILLION, ITS PRESUMPTIONS ARE CAREFULLY FOCUSED ON THREE (3) DISABILITIES: SOFT TISSUE SARCOMAS, CHLORACNE AND PORHYRIA. IN EACH CASE, SUBSTANTIAL EVIDENCE EXISTS RELATING THE DISABILITIES TO EXPOSURE TO 2.4.5-T IN HUMANS.

IN CONSIDERING H.R. 1961 NOW, THIS COMMITTEE WOULD NOT BE PREJUDGING THE OUTCOME OF THE EPIDEMIOLOGICAL STUDIES IT HAS WORKED SO HARD TO ESTABLISH. H.R. 1961 CREATES ONLY AN INTERIM ELIGIBILITY THAT EXPIRES FOLLOWING THE FIRST MAJOR REPORT ON THE EPIDEMIOLOGICAL STUDY MANDATED BY P.L. 96-151. THE COMMITTEE'S HANDS ARE FREE TO ADJUST PRESUMPTIONS TO THE NEW EVIDENCE WHEN IT ARRIVES.

NOR SHOULD THE COMMITTEE FEEL HESITANT ABOUT ACTING IN ADVANCE OF SPECIAL STUDIES. OUR KNOWLEDGE OF 2,4,5-T AND DIOXIN DID NOT BEGIN WITH THE VIETNAM WAR AND IS NOT BOUND BY THE LIMITS OF STUDIES FOCUSED SPECIFICALLY ON VIETNAM VETERANS. THERE IS AN EVER GROWING BODY OF INDEPENDENT ANIMAL AND HUMAN EVIDENCE. AGENT ORANGE POLICY CAN AND MUST REPOND TO THIS MATERIAL AS WELL AS THE SPECIAL STUDIES.

## THE CREDIBILITY OF AGENT ORANGE POLICY

H.R. 1961 WILL PROVIDE COMPENSATION TO SOME 3,000 VIETNAM VETERANS. THAT IS AN IMPORTANT STEP, BUT THE BILL'S SIGNIFICANCE GOES BEYOND THE AID IT OFFERS TO INDIVIDUALS. BY DEMONSTRATING THE CONGRESS' WILLINGNESS TO ACT NOW, H.R. 1961 WILL CONTINUE THE EFFORT BEGUN BY THIS COMMITTEE TO RESTORE CREDIBILITY TO AGENT ORANGE POLICY AND ITS SCIENTIFIC AGENDA.

THE CREDIBILITY OF THE GOVERNMENT'S RESEARCH AGENDA DOES NOT REST EXCLUSIVELY ON THE QUALITY OF ITS SCIENCE. IT ALSO RESTS ON THE INTEGRITY OF THE POLICY IT SERVES. FOR VIETNAM

VETERANS TO TRUST THE CALL FOR MORE RESEARCH, THEY MUST BELIEVE THAT NEW FINDINGS WILL PRODUCE NEW POLICY AND WILL NOT DISAPPEAR BEHIND AN EVER ESCALATING BURDEN OF PROOF AND THE CALL FOR YET MORE RESEARCH. THEY MUST BELIEVE THAT AT SOME POINT ENOUGH WILL BE ENOUGH, AT SOME POINT A LINE WILL BE DRAWN.

REASONABLE PEOPLE CAN DIFFER OVER WHERE THAT LINE SHOULD BE DRAWN, BUT THEY CANNOT DIFFER OVER THE FACT THAT EACH DAY, AS CLAIM AFTER CLAIM IS DENIED, A LINE IS BEING DRAWN IN THE LIFES OF OUR NATION'S VIETNAM VETERANS. IT IS POSSIBLE TO ARGUE THAT THE EVIDENCE SUPPORTING THE DISABILITIES INCLUDED IN H.R. 1961 IS NOT ADEQUATE, BUT IT IS NOT POSSIBLE TO AVOID THE QUESTION THAT MUST FOLLOW UPON THAT ARGUMENT. IF THIS EVIDENCE IS NOT ADEQUATE, THEN HOW TALL A MOUNTAIN OF MATERIAL WILL FINALLY BE REQUIRED.

VIETNAM VETERANS CANNOT BE ASKED TO BELIEVE IN SCIENCE FOR SCIENCE'S SAKE. THEY MUST BE ASKED TO BELIEVE IN SCIENCE IN SERVICE TO A RESPONSIBLE BUT COMPASSIONATE POLICY. H.R. 1961

PROVIDES THAT POLICY. IT DEMONSTRATES THAT THE CONGRESS IS PREPARED TO DRAW A LINE AND ACT.

MR. CHAIRMAN, WITH YOUR PERMISSION I WOULD LIKE TO INSERT IN THE RECORD A COPY OF THE TWO SWEDISH STUDIES, THE OTA AND WORK GROUP REVIEWS OF THOSE STUDIES, AND THE SUBSEQUENT OBSERVATIONS NOTING A RELATIONSHIP BETWEEN EXPOSURE TO 2,4,5-T AND SOFT TISSUE SARCOMAS.

ou

# THE TESTIMONY
# OF THE HONORABLE LEON E. PANETTA
# BEFORE THE
# SUBCOMMITTEE ON COMPENSATION, PENSION, AND INSURANCE
# OF THE
# COMMITTEE ON VETERANS' AFFAIRS

Mr. Chairman, as vice chairman of Vietnam-Era Veterans in Congress (VVIC) and as an original sponsor of H.R. 1961 to provide a presumption of service-connection for specific illnesses appearing in Vietnam veterans, I am honored to have the opportunity to address this subcommittee on the issue of agent orange and the legislation under consideration. I would also like to express my gratitude to VVIC chairman Tom Daschle for introducing H.R. 1961 and to the chairman of this subcommittee, Mr. Douglas Applegate, for convening today's hearing on this important legislation.

It has been over twenty years since the beginning of the American involvement in Vietnam and almost a decade since that involvement ended. Those years were among the most painful and devisive in recent history as Americans confronted profound questions both at home and abroad.

It is perhaps only natural that there remain today a number of unresolved questions stemming from our experiences in Southeast Asia; lingering reminders of the painful era that have long been repressed as we searched as a nation for unity and psychological healing. One such legacy involves the long-term health consequences for U.S. service personnel exposed to chemical herbicides -- including agent orange -- while serving in Vietnam.

As the members of this subcommittee may recall, the practice of herbicide spraying in Vietnam for military purposes was discontinued in early 1971 following the release of a study sponsored by the National Cancer Institute that linked exposure to the herbicides with birth defects in laboratory animals, and the appearance of numerous South Vietnamese newspaper articles which reported health problems among the rural populations sprayed with the defoliants. Concern mounted following the discovery of significant levels of dioxin, a chemcial contaminant considered to be one of the most toxic substances known to man, in agent orange and other phenoxy herbicides sprayed in Vietnam. In all, an estimated 2.8 million American GIs were stationed in Vietnam between 1962 and 1971, the period in which agent orange and other defoliants were used extensively in a campaign to deprive enemy forces of ground cover and food supplies.

In 1979 Congress responded to reports of alarming incidences of serious medical disorders in Vietnam veterans believed to be related to agent orange exposure by directing the Veterans Administration to undertake a study into long-term health effects associated with exposure to phenoxy herbicides (2,4,5-T) and dioxin. The record of what followed has drawn into question the credibility of the VA's agent orange policy. After three years of troubling delays, during which numerous criticisms were leveled at the VA by veterans' service organizations and government officials, the agency reluctantly agreed to transfer the agent orange study to the Center for Disease Control in Atlanta, Georgia. Current estimates are that initial results from the epidemiological study will not be available before 1987.

In a recent report issued by the Veterans Administration, it was reported that 16,564 claims had been filed by veterans for disabilities they believed resulted from exposure to agent orange and other dioxin contaminated herbicides. Of the total, only 1,275 disability claims were allowed, all for reasons other than dioxin poisoning. The VA has as a policy refused to recognize veterans' claims or service-connection for chronic illnesses and other disorders attributed to agent orange except for a rather limited number of cases involving a skin condition known as chloracne. It is the agency oft-stated position that expansion of coverage would be premature in the absence of conclusive medical data to document a cause and effect relationship between exposure to the chemical agents in question and specific health effects other than chloracne.

Before assessing the all important question of causality between exposure to dioxin contaminated herbicides and adverse health effects, we ought to keep in mind that dioxin has been described as one of the most toxic substances every studied -- 150,000 times more toxic than cyanide. While the Veterans Administration steadfastly maintains that it would be imprudent to change its present policy on agent orange prior to completion of the epidemiological study, we must remember the study is designed only to assess the prevalence of cancer and other disorders among Vietnam veterans relative to their peers. It is not, nor should it be, expected that the ongoing study will demonstrate a conclusive medical cause and effect relationship between chemical exposure and adverse health effects.

It is interesting to note that the Veterans Administration already lists a number of disorders it presumes to be service related, despite limited or circumstantial evidence. It is my understanding that the VA today honors veterans' disability claims for multiple sclerosis which are filed up to seven years following discharge from the armed services, rather than the normal one-year limit for filing disability claims. What evidence is there to support the claim that in fact these veterans contracted MS during military service? We know that MS often takes a number of years for its symptoms to manifest themselves, but the fact remains that the VA is willing to accept the presumption in cases involving MS, that the illness was in fact contracted during military service. Sadly, this standard has not been applied in review ing claims filed by Vietnam veterans for disabilities attributed to agent orange exposure.

On the other side of the issue of causality is a growing body of evidence which supports a correlation between exposure to dioxin and 2,4,5-T and increased incidences of certain biological disorders. Specific toxic reactions -- including cancer -- have been observed in laboratory animals following exposure to even minuet concentrations of dioxin. More pertinent to today's discussion, recent medical studies involving agricultural and industrial workers exposed to phenoxy herbicides have documented increased incidences of certain rare illnesses. I commend to the attention of my colleagues a series of Swedish studies which reported a five- to six-fold increased risk of soft-tissue sarcoma in lumberjacks exposed to herbicides containing dioxin. In addition, a National Institute of Occupational Safety and Health (NIOSH) dioxin registry has revealed significantly higher rates of soft-tissue cancers in U.S. industrial workers exposed to the chemical. In fact the VA has on record nearly 1,200 documents relevant to the issue of herbicide toxicity.

Mr. Chairman, I am not in the position to argue the scientific merit of available statistical and medical data relating to dioxin poisoning and 2,4,5-T exposure. I trust the subcommittee will hear expert, albeit undoubtedly conflicting, testimony from members of the scientific community in this matter. What is at question, and focus of the present controversy, is the nature and extent of the government's obligations to gravely ill Vietnam veterans who, while in the service of their country, were exposed to extremely toxic chemical agents.

I do not suggest we abandon reason for the sake of compassion. However, at some point a line must be drawn as to how much evidence must be obtained before action can be taken to assist dying and seriously ill Vietnam veterans. I need only to point to the massive cleanup and relocation effort at Times Beach, Missouri following the discovery in that community of the same deadly dioxin known to have been present in Agent Orange, as evidence that other branches of the federal government recognize the serious health risks associated with dioxin exposure. Can it be that dioxin found in agent orange is any less deadly than that found in Times Beach?

H.R. 1961 is not a reckless response as VA officials would have us believe. The legislation covers three specific disorders, drawn from existing medical data: soft-tissue sarcomas (cancer), porphyria cutanea tarda, and chloracne. In approving this legislation, the Congress would not be prejudging ongoing studies into long-term health effects of exposure to agent orange. H.R. 1961 establishes only an interim eligibility, allowing for review and modification following the conclusion of the epidemiological study. Most importantly, enactment of H.R. 1961 would restore credibility to the VA's agent orange policy and our commitment to compensate veterans for injuries incurred in the service of our country.

Each step of the path toward resovling this issue has seemed interminable. For Vietnam veterans now suffering from cancer or other serious disabilities, the wait must be one of hopeless despair. The call to action is long overdue. Vietnam veterans with cancer are today fighting battles of a war long since over. For these veterans and there families, the battlefields they now face are in corridors of Congress and the Veterans Administration. We as legislators must demonstrate that we are prepared to act by approving H.R..1961 and shifting the burden of proof from the veteran to the VA in awarding service-connected disability claims for illnesses attributable to agent orange.

I thank chairman Applegate and the members of this subcommittee for allowing me the opportunity to participate in this important hearing.

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# CASE-CONTROL STUDY: SOFT-TISSUE SARCOMAS AND EXPOSURE TO PHENOXYACETIC ACIDS OR CHLOROPHENOLS

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**Summary.—In 1977 a number of patients with soft-tissue sarcomas and previous exposure to phenoxyacetic acids were described. Following from these observations a matched case-control study was made. Exposure to chlorophenols was also included in this study. The results showed that exposure to phenoxyacetic acids or chlorophenols gave an approximately 6-fold increase in the risk for this type of tumour. It was not possible to determine, however, whether the carcinogenic effect was exerted by these compounds or by impurities such as chlorinated dibenzodioxins and dibenzofurans that in almost all cases were part of the commercial preparations.**

IN the general debate on environmental hazards in Sweden, few topics have been discussed as vigorously as the phenoxyacetic acids. The debate has focused on their presumptive carcinogenic and teratogenic risks. There are no epidemiological or other reports that have firmly established a correlation between cancer and previous exposure to phenoxyacetic acids in human beings. In an investigation of Swedish railroad workers with exposure to different herbicides, a significantly higher tumour incidence and mortality rate was shown in those with exposure to amitrole, but initially the same findings did not apply to phenoxyacetic acids (Axelson & Sundell, 1974). In a re-analysis of this study doubts were raised about the phenoxyacetic acids as a specific group of products (Axelson & Sundell, 1977). In an accident at the BASF plant, Ludwigshafen, in 1953, 75 persons were exposed to trichlorophenol and chlorinated dioxins. Six malignant neoplasms causing death were found among those exposed, whereas only 3 were expected from comparison with general population data and 4 from comparison with an internal control group (Thiess & Frentzel-Beyme, 1978). Mice with peroral exposure to dioxin over 2 years showed an increased frequency of hepatomas over the control group (Tóth *et al.*, 1978). Another study on mice with peroral exposure to 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) showed an increased tumour incidence in one of two studied strains (Muranyi-Kovacs *et al.*, 1976).

Recently a number of patients with soft-tissue sarcomas and previous heavy exposure to phenoxyacetic acids were reported (Hardell, 1977). This clinical observation resulted in a matched case-control study of this type of tumour. An analysis of exposure to chlorophenols was included in the investigation, as there are related processes in the production of phenoxyacetic acids and chlorophenols, and there may be similar impurities such as chlorinated dibenzodioxins and dibenzofurans in the commercial preparations.

Phenoxy herbicides have been used to control unwanted hardwoods in Swedish forestry since the beginning of the 1950s, usually as a mixture of 2,4-dichlorophenoxyacetic acid (2,4-D) and 2,4,5-T. In order to control mainly aspen, 2,4-D has been used in stump and basal-bark spraying. In farming, 4-chloro-2-methylphenoxyacetic acid (MCPA) has been the dominating herbicide.

Chlorophenols were also introduced in Sweden in the beginning of the 1950s.

About 90% of the consumption has been in sawmills as impregnates or to protect against blue stain in cut and newly sawn timber. Chlorophenols have been used as a fungicide for slime control in the production of paper pulp.

In addition these chemicals have been used as wood preservatives in paints and to waterproof leather and textiles. High levels of chlorinated dibenzodioxins, dibenzofurans and other impurities have been shown in the sawdust from sawmills, especially from the trimming-grading plant where the sawn timber is handled after chlorophenol treatment and drying (Levin *et al.*, 1976).

## MATERIALS AND METHODS

*Cases.*—The studied cases consisted of 21 living and 31 deceased male patients with soft-tissue sarcomas, ranging in age from 26 to 80 years and admitted to the Department of Oncology, University Hospital, Umeå during 1970–77.

*Controls.*—Four matched controls were selected for each case. For each living patient, 3 controls were extracted initially from the National Population Registry matched for sex, age and place of residence. Following a check that these controls were residents of the same municipality as the matched patient at the time of admission to the clinic, a criterion on which a total of 5 controls failed, the 4 ranging closest in age to the patient were used for the study. For dead patients, 10 controls were extracted initially from the National Registry for Causes of Death in order to match each of the deceased patients. They were matched for sex, age, year of death (for 1977, controls from 1976 were used) and place of residence. Deaths from malignant tumours or suicide were excluded. A variation of ±5 years from the age of the patient was accepted because of the small population in some municipalities. If this restriction was not fulfilled, complementary controls from a neighbouring, socio-economically similar municipality were extracted. This was the case for 9 municipalities or 29 controls in total. Dead controls might have been out of work for a long period of time before death and therefore have had less probability of exposure. For this reason their sick leaves and date of retirement were checked through the records of the Public Health Insurance Offices. A criterion was that the controls should have been working until 2 years before the retirement of the patient or, in the case of an unretired patient, until 2 years before his death. Because of this criterion 22 controls were excluded. In addition, one invalid, one person who could not be located and one person without relatives were excluded. As for the living patients, the 4 remaining controls closest in age to the patient were selected for the study.

*Assessment of exposure.*—The investigation started in January 1978. Those persons included in the investigations, for living patients and their controls the persons themselves, and for deceased patients and their controls the next of kin (defined in the following order: wife, children or parents, brother or sister), were if possible contacted by telephone in advance, without the specific nature of the investigation being disclosed. They then received by mail a questionnaire which consisted of a variety of questions about previous and present occupation, different kinds of exposure (especially chemical) in the working environment, smoking habits, etc. Special attention to phenoxyacetic acids or chlorophenols was thereby avoided. The answers were studied and supplemented over the phone by a person not associated with the department, and who did not know whether the interviewed persons were patients or controls. For deceased patients and controls the procedure was the same but in these cases contact was made with the next of kin.

To get as objective information as possible about exposure to phenoxyacetic acids, a questionnaire was also sent to the employers of the persons stating forest work, in order to verify their employment and the use of this chemical. Likewise a questionnaire was sent to sawmills and pulp industries about their use of chlorophenols in the production method.

Low levels of exposure (i.e. a maximum of one day) and late exposure, i.e. 5 years before the tumour was diagnosed, were not included for any of the 2 chemical groups. Sporadic use of impregnating agents during leisure was analysed separately.

*Statistical methods.*—Calculation of relative risk in the matched material was based on principles given by Miettinen (1970). The effect of matching for control of confounding factors was estimated as the quotient of the relative risk in the unmatched to the matched material (*cf.* Miettinen, 1972). The method described by Miettinen (1976) was used in the calculation of the confidence limits.

## RESULTS

The study included 260 persons: 52 patients and 208 controls. Thirty-one patients were deceased. The questionnaire was not answered by 2 controls (0·77%). Twenty-four (46%) of the patients lived in rural areas. According to the 1970 and 1975 census, 26·5% of the population in the age group 25–79 years in the admission region of the Department of Oncology lived in rural areas.

Exposure to phenoxyacetic acids or chlorophenols was registered in 36·5% of the patients and in 9·2% of the controls (Table I). The results obtained from the questionnaire sent to the employer about use of phenoxyacetic acids was uncertain and difficult to evaluate. Records of individual working manuals had not been kept, and the answers were mainly based upon reminiscence. Replies from the employers were obtained for 20/30 persons involved. As to the questionnaire about the use of chlorophenols in sawmills or pulp industry, the answer rate was 97%, and there was a good agreement with the statements given by the examined persons. This indicated that the questionnaires to patients and controls probably also gave reliable information about exposure to phenoxyacetic acids.

TABLE I.—*Exposure to phenoxyacetic acids (Ph) or chlorophenols (Ch) in the matched material (excluding 25 cases where the case and its 4 controls were all negative)*

| No. | Case | Control 1 | Control 2 | Control 3 | Control 4 |
|---|---|---|---|---|---|
| 01 | Ph | — | Ch | — | — |
| 02 | — | Ph | — | — | — |
| 10 | Ch | — | — | — | — |
| 11 | — | — | — | Ph | — |
| 12 | Ch | — | — | — | — |
| 16 | Ph | Ph | — | — | — |
| 18 | Ph | — | — | — | — |
| 19 | Ph | — | — | — | Ph |
| 20 | Ch | Ph | — | — | — |
| 22 | Ph | — | — | — | — |
| 23 | Ph | — | — | — | — |
| 25 | — | — | — | Ch | — |
| 27 | Ph | — | — | — | — |
| 33 | — | — | — | Ch | — |
| 34 | — | — | Ph + Ch | — | — |
| 35 | Ph | — | — | — | — |
| 39 | Ph + Ch | — | — | — | — |
| 43 | Ph | — | — | — | Ph |
| 47 | — | — | Ch | — | — |
| 49 | Ph | — | — | — | Ph |
| 50 | — | Ph | Ch | — | — |
| 51 | Ph | — | — | — | — |
| 52 | Ph | — | — | Ph | — |
| 54 | Ch | Ph | — | — | — |
| 56 | Ch | — | — | — | — |
| 57 | Ch | — | — | — | — |
| 58 | — | Ph | Ph | — | Ph |

The relative risk for soft-tissue sarcomas after exposure to phenoxyacetic acids of chlorophenols was in the matched material 6·2, and after dissolving the matching 5·7 (95% confidence interval 2·9–11·3; Table II). To control the confounding factors the quotient between these relative risks was calculated as 5·7/6·2 = 0·9. This indicates that these confounding factors did not bias the result and the matching was therefore dissolved in the continued analysis.

TABLE II.—*Exposure to phenoxyacetic acids and/or chlorophenols in the total material*

| | Exp. | Non-exp. | Total |
|---|---|---|---|
| Case | 19 | 33 | 52 |
| Control | 19 | 187 | 206 |
| | 38 | 220 | 258 |

Relative risk: 5·7 (95% confidence interval 2·9–11·3).
$\chi^2 = 24\cdot4$.
$P < 0\cdot001$.

As there might be some doubt about the information obtained from relatives compared with that obtained at first hand, a subdivision of the patients and controls was made in the 2 groups of living and deceased. The relative risk for the 21 living patients and their controls was then calculated as 9·9, and for the 31 dead patients and their controls as 3·9. This indicated that second-hand information underestimated the risk.

### *Phenoxyacetic acids*

The effect of exposure to phenoxyacetic acids only was analysed as part of

TABLE III.—*Cases and controls exposed to phenoxyacetic acids. Duration of latency and exposure. Time of exposure within 5 years before the diagnosis has not been included*

| No. | Duration of latency (yrs) | Duration of exposure (months-days) | Chemical | Nature of work |
|---|---|---|---|---|
| Cases | | | | |
| 010 | 15–7 | 18·14 | 2,4,5-T | Work on wet ground |
| 160 | 19 | 00·07 | 2,4-D, 2,4,5-T | Supervisor |
| 120 | 15–13 | 00·07 | MCPA | Tractor-mounted spraying |
| 190 | 27–14 | 40·00 | 2,4-D, 2,4,5-T | Knapsack spraying |
| 220 | 15–8 | 08·00 | 2,4,5-T | Work on wet ground |
| 230 | 20–14 | 03·15 | 2,4-D, 2,4,5-T | Basal-bark spraying, knapsack spraying |
| 270 | 11–0 | 03·21 | 2,4-D, 2,4,5-T | Mistblowing |
| 350 | 13–5 | 05·03 | 2,4-D, 2,4,5-T | Knapsack spraying |
| 390 | 19–9 | 00·02 | 2,4-D (?) | Mistblowing |
| 430 | 20–13 | 03·00 | 2,4-D, 2,4,5-T | Truck-mounted spraying |
| 490 | 10–5 | 03·21 | 2,4-D, 2,4,5-T | Knapsack spraying |
| 510 | 9–3 | 05·00 | 2,4-D, 2,4,5-T | Knapsack spraying |
| 520 | 12–8 | 03·00 | 2,4-D, 2,4,5-T | Basal-bark spraying, knapsack spraying |
| Controls | | | | |
| 021 | 22 | 00·21 | Weedone | Truck-mounted spraying |
| 113 | 17 | 00·06 | 2,4-D, 2,4,5-T | Knapsack spraying |
| 161 | 19–10 | 03·21 | 2,4-D, 2,4,5-T | Knapsack spraying |
| 194 | 24–21 | 02·14 | 2,4-D, 2,4,5-T | Supervisor |
| 201 | 9–6 | 09·00 | 2,4,5-T | Basal-bark spraying, knapsack spraying |
| 342 | 5–0 | 00·05 | MCPA | Tractor-mounted spraying |
| 434 | 20–10 | 05·00 | 2,4-D | Basal-bark spraying |
| 484 | 13–15 | 01·00 | 2,4-D, 2,4,5-T | Knapsack spraying |
| 501 | 17–0 | 08·10 | MCPA, 2,4,5-T | Tractor-mounted spraying, knapsack spraying |
| 523 | 9–1 | 06·00 | 2,4-D, 2,4,5-T | Truck-mounted spraying, basal-bark spraying |
| 541 | 11–10 | 01·00 | 2,4-D, 2,4,5-T | Basal-bark spraying, knapsack spraying |
| 581 | 14–3 | 10·00 | 2,4-D, 2,4,5-T | Basal-bark spraying, knapsack spraying |
| 562 | 12–9 | 04·00 | 2,4-D, 2,4,5-T(?) | Basal-bark spraying, knapsack spraying |
| 584 | 9 | 00·07 | 2,4-D | Basal-bark spraying |

the study. Persons exposed to chlorophenols were excluded, with the exception of one patient and one control with exposure to both chemical groups. Table III gives latent period, duration of exposure and type of work for persons who had used phenoxyacetic acids. The relative risk of exposure to phenoxyacetic acids was calaculated as 5·3 (95% confidence interval, 2·4–11·5; Table IV).

The first report about soft-tissue sarcomas and previous exposure to phenoxyacetic acids was initiated by 3 patients admitted to the Department of Oncology in the autumn of 1976. These findings were followed by a pilot study which disclosed another 4 exposed patients. The relative risk was also calculated excluding the 3 "signal" cases and their controls, in order to check that the risk ratio obtained was not the result of pure chance. One of these patients was considered unexposed throughout the investigation since the next of kin did not confirm any exposure. The relative risk was then calculated as 4·7 (95% confidence interval 2·0–10·7; $\chi^2 = 13{\cdot}2$, $P < 0{\cdot}001$).

TABLE IV.—*Exposure to phenoxyacetic acids only. Patients and controls with exposure to chlorophenols are excluded*

| | Exp. | Non-exp. | Total |
|---|---|---|---|
| Case | 13 | 33 | 46 |
| Control | 14 | 187 | 201 |
| | 27 | 220 | 247 |

Relative risk: 5·3 (2·4–11·5).
$\chi^2 = 17{\cdot}4$.
$P < 0{\cdot}001$.

*Chlorophenols*

When analysing the effect of exposure to chlorophenols, patients and controls exposed to phenoxyacetic acids were excluded, except for one patient and one control with exposure to both chemical groups (Table VI). The calculation gave a relative risk of 6·6. Four patients (7·7%) and 14 controls (6·8%) reported the use of paints containing chlorophenols during their leisure. This implies that such use did not increase the risk for the studied disease. The exposure was in all cases occasional and at a low level. All chlorophenols and 2,4,5-T contain chlorinated dibenzodioxins as an impurity. Only 2 patients and 2 controls had been exposed to preparations not containing dioxins (2,4-D or MCPA) and obviously the observed risk ratios obtained for phenoxy acids and chlorophenols could equally have been caused by dioxins.

TABLE V.—*Exposure to chlorophenols only. Patients and controls exposed to phenoxyacetic acids are excluded*

|  | Exp. | Non-exp. | Total |
|---|---|---|---|
| Case | 7 | 33 | 40 |
| Control | 6 | 187 | 193 |
|  | 13 | 220 | 233 |

RR = 6·6.
$P < 0·001$.

*Dichloro-diphenyl-trichloroethane (DDT)*

The use of DDT-treated plants was common during the 1950s and 1960s, and therefore the potential carcinogenic risk of such exposure was analysed. Two patients and 4 controls with unknown exposure were excluded. Four cases and 14 controls reported exposure to DDT, and the relative risk was then calculated as 1·2.

*Other exposures*

As there might be some other factor related to the occupations in which some people were exposed to the studied chemicals, the risk of the tumour of interest was also calculated for the unexposed persons in these occupations. Seven cases and 47 controls were assigned to that group. The relative risk was 0·6, which implied that the excess risk was related to the chemicals under consideration. Motorized sawing was studied in view of the exhaust fumes and their possible carcinogenic effect. No information was obtained from 2 patients and 2 controls, and they were consequently excluded. Eight cases and 41 controls reported exposure to exhaust fumes, and the relative risk was then calculated as 0·9. Emulsion agents such as diesel oil might have been present in the spraying aggregates. Information about such possible use was obtained from only 10 of the exposed persons. One patient reported such use during one week out of a total exposure of 34 weeks. One patient and 6 controls denied the use of diesel oil in an emulsion and 2 controls confirmed its use. No apparent difference in smoking habits between patients and controls was found. Fifty-one per cent of the patients were smokers (23% ex-smokers) compared with 48% of the controls (22% ex-smokers).

## DISCUSSION

The material consisted of 52 patients with soft-tissue sarcomas. All tumour histologies were reviewed by a pathologist. It is well known that the carcinogenic effect of a specific exposure is often most easily recognized in a rare type of tumour, and also that the aetiological connection in this situation is best documented by a case-control study. In a more common type of tumour, the effect may be blurred by a wider spectrum of exposures and, as regards the rarer tumours, cohort studies often contain too few cases for significant results. In this case a rare type of tumour acted as a signal, and the relation to the specific exposure was first suspected from clinical observations (Hardell, 1977). From a methodological point of view some aspects of the investigation need special comments.

The admission region of the Department of Oncology in Umeå includes the 4 most northern counties in Sweden. All soft-tissue sarcomas in this region are not examined at the department, but any selection of patients regarding possible exposure to phenoxyacetic acids or chlorophenols is unlikely. The probability of exposure to these chemicals is greater in rural areas than in urban areas, and to avoid bias the controls were matched for place of residence.

For dead patients, deceased controls were selected in order to achieve similar conditions when relatives of deceased patients and their matching controls were interviewed. Persons dying from a tumour disease were excluded from these controls, as possible carcinogenicity could be valid also for other types of tumours and thus blur the effect on the studied specific tumour type.

Only general information about the investigation was disclosed in the first telephone contact with the persons concerned and reference to phenoxyacetic acids or chlorophenols was avoided. The questionnaire included a large variety of questions in order to mask the purpose of the investigation. To minimize the risk of biasing the results, the person who made the supplementary interviews did not know whether the interviewed persons were patients or controls. The statements obtained from the questionnaires and the telephone interviews were considered as the single source of information about exposure. The next of kin of one of the first 3 observed patients did not report any exposure to phenoxyacetic acids. In this case the history in the record revealed massive exposure 15–4 years before the diagnosis. This patient was considered unexposed throughout the investigation in order to obtain a conservative estimate of the risk.

### *Confounding*

The method described above for selecting controls excluded confounding factors such as sex, age, place of birth and year of death. Smoking habits were studied and showed no apparent difference between patients and control persons.

Motorized sawing work and related exhaust exposure was analysed, and no significant alteration of the risk could be shown. Theoretically, diesel oil might have been an uncontrolled confounding factor for the effect of phenoxyacetic acids, but hardly so for the chlorophenols. The information obtained was, however, insufficient for a proper evaluation. DDT might also be a confounding factor, but the calculation of relative risk showed no significant increase due to this chemical. The pure substances and impurities could not be evaluated separately as both phenoxyacetic acids and chlorophenols contained impurities such as chlorinated dioxins, dibenzofurans and carriers. Other pesticides might also constitute uncontrolled confounding factors. For example, stump painting with pikloram did occasionally occur during this period. No statements about exposure to pikloram or other such pesticides were obtained by the questionnaires.

### *Conclusion*

The investigation showed an increased risk for soft tissue sarcomas related to the use of phenoxyacetic acids or chlorophenols. It is most unlikely that the results were influenced by uncontrolled confounding factors or other defects in the validity of the study. A specific evaluation of the effect of separate chemical substances was not possible, as nearly all exposed persons were also exposed to chlorinated dioxins, including their most potent form, 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD). The increased risk for this type of tumour after exposure to phenoxyacetic acids or chlorophenols can consequently be caused by the pure chemical substances, impurities in the commercial preparations or a combination of both.

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# CASE-CONTROL STUDY ON MALIGNANT MESENCHYMAL TUMORS OF THE SOFT TISSUE AND EXPOSURE TO CHEMICAL SUBSTANCES

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# CASE-CONTROL SUDY ON MALIGNANT MESENCHYMAL TUMORS OF THE SOFT TISSUE AND EXPOSURE TO CHEMICAL SUBSTANCES

Chemical pesticides have been used in agriculture and forestry in Sweden since the late 1940s. Whereas the use of insecticides has decreased within the past 25 years and the consumption of fungicides has remained approximately constant, the use of herbicides has increased considerably.

Of the 4394 metric tons of pesticide active materials used in agriculture in 1977, 3776 tons (86%) consisted of herbicides. Of the herbicides, phenoxy acids accounted for 2690 tons (71%). In addition to the phenoxy acids being among the most widely used agricultural weed control agents, they are also employed in horticulture and in forestry, both to control broad-leafed weeds and to kill individual trees, i.e. basal bark spraying.

Phenoxy acids have come under vigorous discussion from the environmental viewpoint in recent years, especially in relation to aerial spraying in forestry. The debate primarily concerns the risk to humans.

In a recently conducted case-control study, an indication was obtained of the relationship between malignant mesenchymal tumors of the soft tissue (soft-tissue sarcomas) and exposure to phenoxy acids, with a relative risk of 5.3 (Hardell, Sandstrom 1979). The cases in this study were comprised of patients within the intake area for the oncology clinic of the Regional Hospital at Umea, i.e., the three northernmost counties in Sweden.

Contamination by dibenzofurans and dibenzodioxins, including the highly toxic 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD), should be considered to be present in the preparations with which all of the exposed cases except two came into contact.

The material was also analyzed with regard to exposure to chlorophenols, giving a relative risk of 6.6. Chlorophenols are chemically related to phenoxy acids and may contain similar contaminants in the form of polychlorinated dibenzodioxins (PCDD) and dibenzofurans (PCDF). Chlorophenols have primarily been used to protect against blue stain and as impregnates within the lumber industry, but they have also been employed in wood paints and have been used in tanneries, as proofing agents for fabrics, etc.

The pesticidal use of the phenoxy acid 2,4,5-trichlorophenoxy acetic acid (2,4,5-T) was prohibited in Sweden in 1977, and the phenoxy acids currently available on the market comprise 4-chloro-2-methylphenoxy acetic acid (MCPA) and 2,4-dichlorophenoxy acetic acid (2,4-D), as well as the corresponding phenoxypropionic acids, i.e. mecoprop and dichlorprop. In comparison with 2,4,5-T, these phenoxy acids are less prone to contain impurities in the form of PCDDs and PCDFs, as demonstrated by chemical analysis (Norstrom et al. 1979).

To find out if the earlier findings of a relationship between exposure to phenoxy acids or chlorophenols and soft-tissue sarcomas could be confirmed, a new similar case-referent study of this type of tumor was undertaken. This time, the investigation was performed in southern Sweden, which offered further possibilities to specifically analyze the potential effects of MCPA and 2,4-D, and the analogous phenoxypropionic acids, all of which are widely used in the agricultural area of southern Sweden.

## MATERIALS AND METHODS

The study included only men, and the cases were comprised of patients in the age group of 25-75 years with malignant mesenchymal tumors of the soft tissue (soft-tissue sarcomas) verified by histopathologic confirmation, as diagnosed and reported to the Cancer Registry of the National Social Welfare Board in 1974-78 and who were were living in the five southernmost counties of Sweden (Malmohus, Kristianstad, Blekinge, Kronoberg and Halland) at the time of diagnosis. Both living patients and those who had died were included in the investigations. Controls were selected for each case; deceased controls were used for the deceased cases.

The study was started in February 1979. For every case living at that time, 8 control subjects, matched by community and age, were extracted from the National Population Registry. Out of these, the two control subjects closest in age to each living case were selected, although one individual with severe brain damage was excluded and replaced, along with two who could not be located.

For each deceased case, 10 control subjects matched by community, age and year of death were selected from the National Registry for Causes of Death. For cases who had died in 1978, controls who died in 1977 were used for ethical reasons. Individuals who died of malignant disease were excluded as unsuitable for incorporation in the control group (primary relationship to exposure possible: see Axelson 1979a). Because of considerations pertaining to the ethics of interviewing, individuals whose death was due to suicide were also excluded. In both of these instances, excluded individuals were replaced by the next nearest individuals in age.

Deviations of more than 5 years in age between cases and controls were not acceptable; in some cases, because of the small populations in certain communities, it was necessary to extract 12 additional control subjects from nearby, socio-economically similar communities.

In order to avoid misleading results due to the fact that the deceased controls may not have worked for a long time before death and thus would have had a lower possibility for occupational exposures of various types, their disability or pensioning histories were checked by consulting Public Health Insurance records. The controls were accepted only if they had been working until 5 years before the pensioning or death of the cases. As a result of this requirement, 17 initially selected deceased controls were removed and replaced. Also removed and replaced was one deceased control subject who was an immigrant and whose relatives could not be located.

In the same manner as for the living patients, the two remaining deceased controls nearest in age were selected for the deceased patients. The closest relatives in the case of the deceased individuals were defined in succession as spouse, child or parent, sibling, other.

## ASSESSMENT OF EXPOSURE

Whenever possible, the individuals included in the investigation were contacted in advance by telephone, but without disclosing the issues of primary importance for the investigation. After this they received a questionnaire with a number of questions regarding previous and current occupation, exposure conditions in the occupational environment, smoking habits, etc. Special attention directed toward phenoxy acids or chlorophenols was thereby avoided.

All replies were checked and completed, if necessary by telephone, by an individual who did not know whether the person being interviewed was a case or a control. Any incomplete or unclear answers to the questions, especially concerning exposure to solvents and biocides, were then supplemented.

In addition, all individuals who reported having worked in agriculture, forestry or horticulture during the time when the biocides were used in Sweden were contacted. In this manner a uniform evaluation of the exposure conditions for this occupational category was obtained, in terms of the biocides in question as well as other substances and working processes.

In some cases the data in the questionnaire were incomplete, e.g., regarding sprayings carried out by machine pool stations, and the situation was then clarified by telephone interviews. If the subject did not have a telephone, the interviews were completed by mail.

For deceased individuals, the procedure was the same, except that naturally the closest relative (defined as above) was the target of the questionnaire and the telephone contacts.

The interviewers also made contact with the employer, agricultural board members, machine pool stations, neighbors and others in order to clarify the possibility of exposure. As was mentioned above, this was done without knowledge of whether each subject was a case or a control.

For the subjects who themselves did not participate in the spraying, but who were employed, e.g., in a machine pool station, as described in the following, a separate analysis was carried out to detect any possible increase in risk for the tumor type in question. Low-grade exposure to phenoxy acids, defined as exposure for a total period of time less than one day, was disregarded. In the case of spraying of rights-of-way on the Swedish Railway, amitrole was primarily used, but other pesticides including phenoxy acids were also used. However, it was not possible to establish with certainty which preparation types were used by contacting the subjects themselves, their co-workers, or their employers, and therefore railway workers who reported pesticide exposure were regarded as unexposed to phenoxy acids. This applied to one case and one control.

Since it is known that blood lipid lowering drugs of the clofibrate type are derived from a phenoxy acid, attention was likewise directed in the investigation toward the presence of such medications. For this reason, medical records were also examined. However, no one fulfilled the latency criteria as defined below, and therefore no analysis of the material in this regard was carried out.

A separate analysis was carried out for chlorophenol-containing lumber protection agents with regard to low-grade exposure, which was defined as a maximum of one continuous week of exposure or repeated short-term exposures totaling a month at most. Exposure to chlorophenols may also have occurred among individuals in contact with cutting oils and among employees in the shoe and leatherware industries, but since it is difficult to assess such exposure accurately, these groups were considered nonexposed. On the other hand, one case employed in a tannery was adjudged to be exposed to chlorophenols in view of the frequent exposure inherent in such activity.

The latency time for chemical induction of malignant solid tumors in man is generally considered to be very long, and on the average in the range of 15-30 years (Hueper, Conway 1964). Therefore, regarding both phenoxy acids and chlorophenols, recent exposure of either cases or corresponding controls, defined as exposure within a five-year period prior to diagnosis of the case, was excluded in the analysis.

The material was also analyzed with regard to exposure to other agents, e.g., organic solvents, DDT and mercury-containing seed dressing agents, since these can have a confounding effect, which may often be the case for simultaneous or consecutive occupational exposures, insofar as they comprise risk factors for the diseases in question (see Axelson 1978, 1979b).

It should also be added that detailed knowledge on dispersion agents is lacking, and that the various preparations are thus referred to in terms of the active ingredients.

## STATISTICAL METHODS

Calculations of the chi-square values and the relative risk in the matched material were based on the principles of Miettinen (1969, 1970). The effect of retaining versus dissolving the matching was evaluated as the quotient of the relative risk in the unmatched to the matched material (see Miettinen 1972). A test-based, approximative method was used in calculation of the confidence intervals (Miettinen 1976).

## RESULTS

The material included a total of 329 subjects, 110 of them cases and 219 controls. Thirty-eight of the cases were deceased. The shortfall in the study population consisted of one control (0.3%), considered to be unexposed in calculations with matching retained, and excluded in the calculations after matching was dissolved. Exposure to phenoxy acids or chlorophenols was reported by 22.7% of the cases and 5.9% of the controls. One case and one control with less than one day of exposure were considered unexposed (employed in their own gardens).

The relative risk of malignant mesenchymal tumors of the soft tissue after exposure to phenoxy acids or chlorophenols was found to be 5.1 in the matched material (see Table 1), and after dissolving of matching 4.7. The effect of matching is illustrated by the ratio between these relative risks, i.e., 4.7/5.1 = 0.92; this ratio of less than 1.0 indicated that the matching factors rather masked the risk, and therefore dissolving of matching seemed acceptable for further analysis, which was thus considerably simplified.

Table 1

EXPOSURE TO PHENOXY ACIDS OR CHLOROPHENOLS WITHIN TRIPLETS (CASE AND TWO CONTROLS)

| Case with exposure to phenoxy acids or chlorophenols | No. of controls with exposure to phenoxy acids or chlorophenols | | |
|---|---|---|---|
| | 2 | 1 | 0 |
| Yes | 0 | 4 | 21 |
| No | 0 | 9 | 76 |

Relative risk = 5.1; $X^2(1) = 20.1$
95% confidence interval = 2.5-10.4

Table 2

EXPOSURE TO PHENOXY ACIDS IN CASES AND CONTROLS WHEN THOSE EXPOSED TO CHLOROPHENOLS WERE EXCLUDED AND MATCHING WAS DISSOLVED

| | Exposed | | Unexposed |
|---|---|---|---|
| | More than 30 days | 30 days or less | |
| Test cases | 7 | 7 | 85 |
| Controls | 2 | 3 | 206 |
| Relative risk | 8.5 | 5.7 | (1.0) |

For exposed compared to unexposed individuals, one obtains relative risk = 6.8; $X^2(1) = 16.2$.
95% confidence interval = 2.6-17.3

PHENOXY ACIDS

In a partial study the effect of exposure to phenoxy acids alone was analyzed.

All individuals exposed to chlorophenois were excluded from this partial study. The relative risk for exposure to phenoxy acids amounted to 6.8 (Table 2), and an imitative but nonsignificant dose-response relationship was obtained upon subdivision of the material.

In order to clarify whether exposure to phenoxy acids without contamination by PCDDs or PCDFs could be associated with the tumor types investigated, a special analysis was undertaken excluding individuals exposed to 2,4,5,T. The relative risk upon exposure to the other phenoxy acids (MCPA, 2,4-D, mecoprop, dichlorprop) amounted to 4.2 (Table 3).

Eight cases (7.3%) and 17 controls (7.8%) stated that they had worked or were now working in agriculture where chemical weed control was carried out by a machine pool station or other persons, without their own active participation in in the work.

Table 3

EXPOSURE TO THE PHENOXY ACIDS MCPA, 2,4,-D, MECOPROP AND DICHLORPROP WHEN INDIVIDUALS EXPOSED TO 2,4,5-T AND CHLOROPHENOLS WERE EXCLUDED AND MATCHING WAS DISSOLVED

| | Exposed | Unexposed |
|---|---|---|
| Cases | 7 | 85 |
| Controls | 4 | 206 |

Relative risk = 4.2 $X^2(1) = 5.9$

95% confidence interval = 1.3-13.4

EXPOSURE TO CHLOROPHENOLS IN CASES AND CONTROLS WHEN INDIVIDUALS EXPOSED TO PHENOXY ACIDS WERE EXCLUDED AND MATCHING WAS DISSOLVED

| | Exposed | Unexposed |
|---|---|---|
| Cases | 11 | 85 |
| Controls | 8 | 206 |

Relative risk = 3.3· $X^2(1)$ = 6.8

95% confidence interval = 1.3-8.1

CHLOROPHENOLS

Analysis of exposure to chlorophenols, with exclusion of the subjects exposed to phenoxy acids, gave a relative risk of 3.3 (Table 4). Low-grade exposure was present in 7.3% of the cases and 5.5% of the controls, and thus apparently did not bring about an increase in the risk.

OTHER EXPOSURE

The relative risk was also calculated for exposure to organic solvents. High- and low-grade exposures were defined with the same time criteria applied to exposure to chlorophenols (see the preceding); this resulted in a relative risk of 1.6 for high grade exposure and 1.9 for low-grade exposure, both with a 90% confidence interval encompassing 1.0. The questionnaire also included a survey of exposure to various other substances and materials (see Table 5).

Data on exposure to asbestos, glass fibers and power saws are more uncertain then those for the other exposure conditions, since the replies to the questionnaires in these cases were used as the only information, without telephone supplementation of uncertain data. As concerns the relative risks for exposure

Table 5

FREQUENCY AND EXPOSURE IN PERCENT TO VARIOUS AGENTS, ETC., IN 110 CASES AND 219 CONTROLS IN THE TOTAL MATERIAL, OR 95 CASES AND 212 CONTROLS AFTER EXCLUDING THOSE EXPOSED TO PHENOXY ACIDS OR CHLOROPHENOLS. SEE OTHER TABLES WITH REFERENCE TO EXPOSURE TO PHENOXY ACIDS AND CHLOROPHENOLS.

| | Exposure frequency, % | |
|---|---|---|
| Agent, etc. | Case | Controls |
| Total material | (110 cases) | (219 controls) |
| Asbestos | 4.5 | 3.7 |
| Glass fiber | 7.3 | 3.7 |
| Power saws | 8.2 | 5.5 |
| Smoking, now or previously | 71.8 | 70.3 |
| Pesticides | | |
| - total* | 21.8 | 11.0 |
| - amitrole | 0.9 | 0.5 |
| - bromophos | 1.8 | 0.9 |
| - DDT | 6.4 | 5.0 |
| - dinoseb | 2.7 | 0.9 |
| - fenitrothion | 0.9 | 0.5 |
| - Hg-containing seed dressings | 8.2 | 4.6 |
| - lindane | 0.9 | 0.5 |
| - Na chlorate | 0.9 | 0.9 |
| - nicotine | 2.7 | 0.5 |
| - other** | 13.6 | 6.8 |
| Materials after exclusion | (95 cases) | (212 controls) |
| Pesticides<br>-total* | 9.5 | 8.0 |

*No. of individuals exposed to one or more pesticides (other than phenoxy acids or chlorophenols)

**Includes, among other things, pesticides in which either cases or controls were unrepresented (a maximum of 2 in either group is present, while the other is unrepresented), as well as unknown preparations (4 cases and 6 controls); 15 different preparations were mentioned in this group.

to DDT, mercury-containing seed dressings, etc., according to Table 5, these were not definitely elevated, since the 90% confidence interval for the relative risk for each preparation included 1.0.

## DISCUSSION

The investigation was undertaken because of the previously demonstrated association between malignant mesenchymal tumors of the soft tissue (soft-tissue sarcomas) and exposure to phenoxy acids and chlorophenols. The aim of this new study was partly to determine whether the previous investigation results could be reproduced in a different geographic area, and partly to clarify whether the exposures, currently occuring in agricultural practice, to phenoxy acids free from PCDDs and PCDFs might also be associated with this particular group of tumors. The tumor slides from each patient were rechecked by two pathologists (Berg, Angervall), and as a result, 11 of the cases initially extracted from the Cancer Registry were eliminated bacause of incorrect or uncertain diagnosis. The histopathologic distributions of the tumor cases are shown in Table 6.

Table 6

HISTOPATHOLOGIC DIAGNOSIS IN THE REMAINING MATERIAL

| Diagnosis | No. of Cases |
|---|---|
| Leiomyosarcoma | 33 |
| Malignant fibrous histiocytoma | 19 |
| Liposarcoma | 15 |
| Neurogenic sarcoma | 11 |
| Angiosarcoma | 9 |
| Myxofibrosarcoma | 7 |
| Fibrosarcoma | 5 |
| Dermatofibrosarcoma | 3 |
| Atypical fibroxanthoma | 2 |
| Synovial sarcoma | 2 |
| Sarcoma, not further specified | 2 |
| Ewing sacroma (extraskeletal) | 1 |
| Rhabdomyosarcoma | 1 |
| Total | 110 |

In carrying out this study, the same basic methodology was used as in the corresponding previous study. Deceased individuals were selected as controls for deceased patients in order to obtain similar circumstances for the assessment of exposure. As was mentioned in the section on materials and methods, however, deceased individuals with malignant tumors were not included among the controls, since possible carcinogenicity of the studied exposures might also lead to other forms of tumor mortality. For living control subjects this restriction was not applied, and the occurrence of possible tumor diseases was not questioned. Therefore, in this regard the study can be considered to be biased in the conservative direction. One living control subject with massive exposure to MCPA also spontaneously reported in his questionnaire that he was undergoing treatment for a malignant tumor.

The questionnaire used in the previous studies in northern Sweden served as the basis for the questionnaire used in the new study. A large number of occupational-related questions were used to mask the intention of the investigation, and the supplementary interviews were carried out without knowledge of the status of the individual as a case or a control.

Potential problems of confounding relating to age, sex, residence and year of death were avoided by restriction of the material and by matching. Exposures to pesticides other than phenoxy acids might be imagined to constitute risk factors for the diseases investigated and thus to exert a confounding effect, since the individuals using phenoxy acids were also frequently in contact with other preparations used for the control of weeds, insects or fungus diseases. Thus, there tends to be a co-variation in exposure, which makes it difficult to fully evaluate the effect of simultaneous or consecutive exposures to different pesticides. Similar considerations also apply to the dispersion agents and possible unknown contaminants.

The preparations used, in addition to phenoxy acids, in agriculture, forestry and horticulture could frequently be described by the subjects, but each individual preparation type occurred only sporadically (Table 5).

On the whole, an overrepresentation of exposure existed among the cases for pesticides other than phenoxy acids. However, if the individuals in the case and control groups who reported simultaneous exposure to phenoxy acids were excluded, 9.5% of the remaining cases and 8.0% of the remaining controls were exposed to various other pesticides, which thus do not seem to exert any demonstrable effect. No risk calculation for individual pesticides other than phenoxy acids seemed reasonable because of the small numbers of exposed cases and controls.

When exposure data are collected via questionnaires and interviews, a certain possibility exists that the cases will have a greater interest in the questions than the healthy controls do. In order to detect any distortion of this kind, the material was also analyzed with regard to exposure to phenoxy acids among cases and controls employed in agriculture or forestry. This gave, for exposed individuals in forestry/ agriculture, a relative risk of 5.7 compared to other occupations without exposure, while unexposed individuals within agriculture and forestry had a relative risk of 0.9 (Table 7). If the exposure was significantly overestimated among the cases and underestimated among the controls, a value of far less than 1.0 would have been obtained in calculating the relative risk among unexposed individuals in agriculture and forestry. The observed relative risk of 0.9 indicates that no substantial distortion in the assessment of exposure to phenoxy acids occurred in the study. Chlorophenol-exposed individuals were included in this analysis.

Table 7

EXPOSURE TO PHENOXY ACIDS IN CASES AND CONTROLS DIVIDED ACCORDING TO OCCUPATION

| | Agriculture/forestry | | Other occupations | |
|---|---|---|---|---|
| | Exposed | Unexposed | Exposed | Unexposed |
| Cases | 13 | 18 | 1 | 78 |
| Controls | 5 | 42 | 0 | 172 |
| Relative risk | 5.7 | 0.9 | | (1.0) |

In summary, this study indicates that exposure to phenoxy acids may constitute a risk factor in the development of malignant mesenchymal tumors of the soft tissue, and that the risk is not limited only to the phenoxy acids which, like certain chlorophenols, can contain polychlorinated dibenzodioxins and dibenzofurans, but also to other phenoxy acids.

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OFFICE OF TECHNOLOGY ASSESSMENT,
*Washington, D.C., June 26, 1980.*

Hon. ALAN CRANSTON,
*Chairman, Senate Committee on Veterans' Affairs, U.S. Senate, Washington, D.C.*

DEAR CHAIRMAN CRANSTON: Thank you for your letter of May 22, 1980, concerning five epidemiologic studies of phenoxyacid herbicides. Michael Gough of the Office of Technology Assessment Health Program had earlier reviewed these papers on an informal basis in response to a request from your committee staff. To obtain an expert outside review of the papers, we asked Dr. Richard Remington, Dean of the School of Public Health, University of Michigan to read and comment on the papers Dean Remington has, as you know, agreed to serve as chairman of the OTA Advisory Panel for the review of the VA Epidemiologic Study of Viet Nam Veterans.

Dean Remington's letter to Michael Gough and his review of the five papers are attached. I wish to bring your attention to two points made in the letter. The first is the high marks accorded to the three case-control studies carried out by the Swedish workers. Dean Remington cites these as "... among the most carefully conducted investigations of their type that I have ever seen. *In toto*, the Swedish work is credible if not fully conclusive." I would add a further reservation, because all of the Swedish case-control studies have some authors in common, a possibility exists that some unconscious, undetected bias colors all the studies. This is not to suggest that the work is less than excellent, but only to point out that verification of these studies by an independent investigation would remove that reservation. I understand that the Interagency Work Group on Phenoxyacid Herbicides has also concluded that the Swedish case-control studies have merit. Taken all together, these studies do suggest that health effects have resulted from exposure to phenoxyacid herbicides in forestry work.

The other point made by Dean Remington is that he now thinks "... that it would be possible using combined military experience to design a useful study of reasonable sensitivity ..." He refers, of course, to the proposed VA study.

Thank you for this opportunity to aid you in your efforts to keep abreast of developments in this difficult area.

Sincerely yours,

JOYCE C. LASHOF, M.D.,
*Assistant Director.*

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THE UNIVERSITY OF MICHIGAN,
*Ann Arbor, Mich., June 19, 1980.*

Dr. MICHAEL GOUGH,
*Project Director, Office of Technology Assessment, Congress of the United States, Washington, D.C.*

DEAR MIKE: I have now been able to review the five investigations into possible relationships between Agent Orange and human health and disease. My detailed critique of the individual studies is attached to this letter. Two of the studies are cohort or prospective investigations. The first of these involving the BASF workers exposed in 1953 is almost totally unhelpful. I do not believe it is even worth citing as evidence in one direction or the other. However, the four Swedish studies are generally good and do considerably raise the index of suspicion concerning possible carcinogenicity of phenoxy acids. The prospective study of Axelson et al. is small and suffers from some analytical difficulties. The case control investigations are among the most carefully conducted investigations of their type that I have ever seen. In toto, the Swedish work is credible if not fully conclusive. Certainly this work would seem to justify further investigation.

I have also been able to review the critique by the NAS panel of the proposed Air Force "ranch hand" investigation. The critique is good and I generally agree with the views of the majority. However, Dr. Kurland raises important points in his minority report. I must frankly admit that my level of optimism concerning the possible utility of prospective epidemiologic investigations in this area has increased since studying these documents. I now believe that it would be possible using combined military experience to design a useful study of reasonable sensitivity to important health effects.

Thank you for your patience. See you soon.

Best personal regards.

Sincerely,

RICHARD D. REMINGTON, Ph. D.,
Dean.

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## SPECIFIC SUMMARY CRITIQUE OF FIVE INVESTIGATIONS RELATED TO CONCERNS ABOUT AGENT ORANGE

(By Richard D. Remington)

1. "Mortality Study of Persons Exposed to Dioxin Following an Accident Which Occurred in the BASF on 13 November, 1953" by Thiess and Frentzel-Beyme.

This study reports on an average 20-year follow-up experience of 75 employees of the BASF in West Germany in 1953. A matched control group and three external control series, based on local and regional vital statistics were selected. The study purports to show an increased frequency of stomach cancer in the dioxin-exposed group.

Unfortunately, both the report and the investigation itself are inadequate and shed little light on the important questions under investigation. The case series is small and the risk of cancer of specific sites even over so long a follow-up period remains at a low level. Indeed, the number of stomach cancer cases in the exposed group is only four. All cause mortality in the exposed group is lower than that of one external comparison group and not different from the other two groups. It is somewhat higher than that of the internal control group. Inadequacies in the description of the investigation include (1) no age breakdown of the exposed population; (2) no description of the matching procedure beyond the global statement that matching by age and date of entry into the factory took place. Was the age matching to a single year, for example, or only to five-year age groups? Within what limits was date of entry controlled? (3) The comparison group is stated to have been selected "at random from the universe of all persons ever included in a cohort study until now." This description is unclear and inadequate. Did a formal randomization occur, i.e., using random number tables?

The authors select stomach cancer for particular attention, and this may not be unreasonable. However, they ignore other differences, for example, among violent deaths. Comparisons between mortality structure by cause with all four control groups reveals substantial variation. In fact, inspection of differences among the external control groups reveals some very large discrepancies, for example, in cardiovascular diseases, overall natural deaths, and all cause mortality rates. These differences themselves cast doubt upon other comparisons within the investigation.

Finally, tests of significance using the Poisson distribution produce estimated p values for the study. This is a "default" type of analysis. The authors seem to believe that use of the Poisson distribution is justified whenever one is inspecting small numbers of events. Nothing could be farther from the truth. The homogeneity assumption for individual event probabilities is almost certainly violated in these data, involving as they do a broad range of ages and other basic characteristics. This lack of homogeneity might well induce an appropriate model whose true variance is well in excess of that of the Poisson distribution.

In summary, this investigation really does not bear centrally on the question of Agent Orange's possible carcinogenicity. The study is inadequately described. The experience is limited. The analytical techniques are questionable. Thus, this investigation neither establishes nor excludes a possible association between dioxin and stomach cancer (or any other cause of death).

2. "Herbicide Exposure and Tumor Mortality: An Updated Epidemiological Investigation on Swedish Railroad Workers" by Axelson, Sundell, Andersson, Edling, Hogstedt, and Kling.

This updated epidemiologic investigation by Swedish railroad workers exposed to herbicides has been carefully conducted and is well described in the research report. A cohort of 348 individuals were followed through October 1978. Mortality ascertainment is very complete and the follow-up experience is excellent. The authors have included only individuals exposed to herbicides for more than 45 days during the period 1957 through 1972. This again is a strength of the investigation. In addition, they have subdivided exposure to amitrol from that to phenoxy acids, again a strength. Statistical significance is once again assessed by the Poisson distribution and earlier reservations apply here. Thus, estimated p values can only be taken as approximations.

The results of this investigation are suggestive. Overall tumor mortality to workers exposed to phenoxy acids appears to have been increased as does mortality due to stomach cancer. This would appear particularly to be true for workers exposed in the early years, although here the numbers appear to be inadequate to provide a breakdown into amitrol and phenoxy acid subgroups.

Once again, the numbers available to this study are inadequate to permit definite conclusions. However, the results of this careful investigation must raise the index of suspicion concerning possible carcinogenicity of phenoxy acids.

3. "Case-Control Study: Soft-Tissue Sarcomas and Exposure to Phenoxyacetic Acids or Chlorophenols" by Hardell and Sandstrom.

This case control study compares 52 living or deceased male patients with soft-tissue sarcomas admitted to the Department of Oncology in Umea, Sweden in 1970–77 with carefully selected matched controls. Aside from the intrinsic limitations of case control methodology, this is an excellent investigation. The authors are unusually careful to exclude questionable cases and controls and to detect and eliminate obvious sources of bias. Thus, their finding of a relative risk of 5.3 for exposure to phenoxyacetic acids must be taken seriously. Their statistical procedures are modern and carefully selected. Their attention to detail is exemplary.

The only misgiving concerning the study is a generic one. Case control studies are uniquely susceptible to hidden sources of bias of ascertainment, recall, and patient selection. The methodology, no matter how careful, cannot guarantee the absence of such biases. Even so, the findings of this particular investigation are suggestive and serve further to raise the index of suspicion concerning induction of soft-tissue sarcoma by exposure to phenoxyacetic acids.

4. "Case-Control Study on Malignant Mesenchymal Tumors of the Soft Tissue and Exposure to Chemical Substances" by Eriksson, Hardell, Berg, Moller, and Axelson.

This study is similar to No. 3 above but concentrates on experience in Southern Sweden. The results are generally similar to that of the earlier investigation and the methods virtually identical. Again, the results are consistent with the hypothesis that phenoxy acid exposure increases the risk of tumors of this type. Once again, the investigators have exercised unusual care to exclude biasing effects as far as they are able to do so. However, again, case control methodology is intrinsically susceptible to subtle and unmeasurable biases.

5. "Malignant Lymphoma and Exposure to Chemical Substances, Especially Organic Solvents, Chlorophenols and Phenoxy Acids" by Hardell, Eriksson and Lenner.

This case control study is by the same investigative unit responsible for references 3 and 4 above. This time a case control study of patients with malignant lymphoma was conducted using methodology similar to that in the earlier investigations of soft tissue sarcoma. Again, a substantial and statistically significant relative risk is found for this group of tumors. And again, phenoxy acid exposure is specifically incriminated. Earlier comments concerning the care these investigators have applied to the study are also appropriate here, and similar limitations of case control methods should be noted.

NATIONAL TOXICOLOGY PROGRAM

Chair, Interagency Work Group on Phenoxy Herbicides DATE: June 25, 1980
and Contaminants

Scientific Panel, IWG

CT: Evaluation of Five Scientific Papers on the Carcinogenicity of Chemicals that were Constituents of Agent Orange

The Scientific Panel is in receipt of 4 Swedish and 1 German paper. They are:

1. L. Hardell and A. Sandstrom. Case Control Study: Soft Tissue Sarcomas and Exposure to Phenoxy Acetic Acids or Chlorophenols. British Journal of Cancer 39: 711-717 (1979).
2. M. Eriksson, L. Hardell, N. O. Berg, T. Moler, and O. Axelson. Case Control Study on Malignant Mesenchymal Tumors of the Soft Tissue and Exposure to Chemical Substances. Lakartidningen 76: 3872-3875 (1979). (EPA Translation)
3. L. Hardell, M. Eriksson and P. Lenner. Malignant Lymphoma and Exposure to Chemical Substances, Especially Organic Solvents, Chlorophenols and Phenoxy Acids. Lakartidningen 77(4): 208-210 (1980). (EPA Translation)
4. O. Axelson, L. Sundell, K. Andersson, C. Edling, C. Hogstedt, and H. Kling. Herbicide Exposure and Tumor Mortality; An Updated Epidemiological Investigation on Swedish Railroad Workers (Manuscript form 1980).
5. A. M. Thiess and R. Frentzel-Beyme. Mortality Study of Persons Exposed to Dioxin Following an Accident which Occurred in the BASF on November 13, 1953. Presented at the Fifth International Conference on Medichem, San Francisco, California, September 1977.

## Evaluation

Papers Nos. 1, 2, and 3 have a common design with L. Hardell appearing as first or second author. Each of the three studies appear to have been well executed although fairly permissive exposure criteria were utilized. Of particular interest to the Scientific Panel are the analyses which the authors defined as exposure only to phenoxy acid herbicides which identified a relative risk for soft tissue sarcoma of 4.3 (paper #1) or 6.8 (paper #2); and for malignant lymphoma 4.8 (paper #3). The phenoxy acid exposures in paper #1 are reported to be with 2,4,5-T and 2,4-D; thus the possible role of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) cannot be discounted. In paper #2 the authors suggested that the increased risk may also be associated with phenoxy acids that do not contain TCDD. Paper #3 did not present separate data as a function of exposure to phenoxy acids with or without the TCDD contaminant.

The similarity of design and involvement of at least one investigator in all three instances could permit the recurrence of an "unobserved bias" which weakens the Panel's acceptance that studies #1 and #2 represent a true independent verification of the findings.

In spite of the reservations that are generally associated with these case control epidemiology studies, i.e., permissive criteria for establishing "exposure" which varied between the studies; memory bias by patients or relatives that there was "exposure" because of a traumatic event such as cancer, the studies show a correlation between exposure to phenoxy acid herbicides and an increased risk of some forms of cancer. Independent verification would further validate these studies.

Paper #4 represents 348 persons which is small for this type of mortality study. The authors reported that the observed number of tumor deaths is higher than expected and that the causal relationship to specific agents (amitrol and phenoxy acids) are unclear. The interpretation of three stomach cancers is very tenuous due to the size of the population and the possible bias of familial or genetic relationship.

Paper #5 represents a study of 75 workers which should be considered as a clinical observation. Genetic or familial association of the three stomach carcinomas needs to be ascertained.

The full utility of small populations such as are represented in papers #4 and #5 can best be realized through the development of an International Registry which includes a number of such populations where the statistical power of such analyses can be substantially enhanced. The development of such a Registry is being actively pursued.

John A. Moore, D.V.M.
Chair, Scientific Panel

# THE LANCET

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NEWSPAPER

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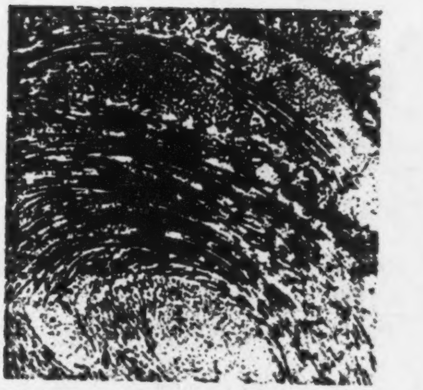

Fig. 1—In 10th week of pregnancy the cervical tissue consists of dense lattice of collagen bundles with sparse amount of ground substance (14 500).

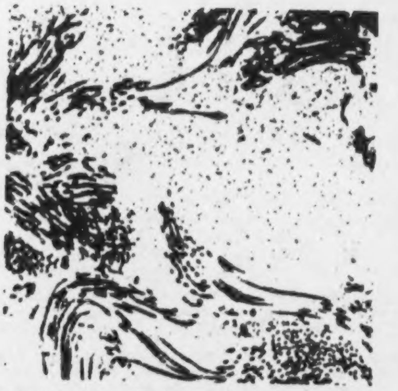

Fig. 2—15 h after gel application the collagen fibres are split up and separated at least partly due to an increase in the amount of ground substance (11 500).

of the cervix before and during labour has been considered "one of the most pressing problems in obstetrics".[3]

Intracervical application of a small dose of prostaglandin $E_2$ ($PGE_2$) in gel ripens the cervix both before termination of early pregnancy by dilatation and evacuation and before induction of term labour.[4,5] Since cervical priming has been observed also in patients without prominent myometrial activity,[6,7] it might be asked which changes of the cervical ultrastructure are responsible for the ripening process. We describe here morphological and biochemical changes of the human pregnant cervix after $PGE_2$ induced cervical ripening.

Biopsies of the cervix were done immediately before and 15 h after intracervical application of 0·5 mg $PGE_2$ gel in twenty nulliparous patients admitted for termination of pregnancy late in the first trimester. The mucosa was removed and the samples were examined by electron microscopy and analysed for sulphated glycosaminoglycans and water.

The biopsy material examined morphologically was fixed in 2·5% glutaraldehyde in 0·2 mol/l phosphate buffer for 1-2 h. It was then rinsed and post-fixed in 1% osmium tetroxide in the same buffer and dehydrated and embedded in 'Vestopal W'. Semi-thin sections for orientation, ultrathin sections for electron microscopy were cut on a LKB 'Ultratome III' and examined in a Philips EM 300.

The material for biochemical analysis was weighed and immersed in liquid nitrogen within 3 min of sampling. The dry weight was determined after heating the samples at 110°C in vacuum over phosphorous pentoxide for 5 h. After delipidation with acetone the material was digested with papain and then subjected to ion exchange chromatography on DE-52-cellulose. The sulphated glycosaminoglycans were eluted with 3 mol/l pyridine acetate and quantitated by hexosamine analysis by the Elson-Morgan reaction after hydrolysis.[8]

The results of the morphological investigations are summarised in two figures. The cervical stroma in early pregnancy consisted of tightly woven bundles of collagenous fibres separated by a sparse amount of ground substance (fig. 1). After $PGE_2$ gel treatment a remarkable change in cervical ultrastructure ensued (fig. 2). The collagenous fibres were more or less separated, split up, or dissolved whereas the amount of ground substance seemed to be considerably increased. Moreover, the fibroblasts were enriched in vesicles localised close to the plasma membrane. The concentration of sulphated glycosaminoglycans increased by almost 20% 15 h after $PGE_2$ gel application. The water content remained unchanged as did the total amount of collagen, as estimated by the amount of hydroxyproline.

Thus intracervical application of $PGE_2$, both morphologically and biochemically, seems to induce a prominent rebuilding of the cervical connective tissue.

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## 2,4,5-T, TRICHLOROPHENOL, AND SOFT TISSUE SARCOMA

SIR,—Case-control studies from Sweden demonstrate relative risks of 6·2[1] and 5·1[2] for occupational exposure to phenoxy herbicides and chlorophenols among cases of soft tissue sarcoma. To estimate whether this excess risk can be detected among workers in the United States, we reviewed reports of four exposed cohorts (refs 3-5 and J. A. Zack unpublished) studied by epidemiologists from Dow Chemical U.S.A. and the Monsanto Company. Of the 105 deaths reported in the four cohorts, 3 (2·9%) were due to soft tissue sarcoma. In rough comparison, for 1975 only 0·07% of deaths in U.S. males aged 20–84 years were due to soft tissue sarcoma (ICD 171, 8th revision).[6]

The workers from the four U.S. cohorts were exposed either to the phenoxy herbicide 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) or to 2,4,5-trichlorophenol (TCP), a feedstock for 2,4,5-T. Both 2,4,5-T and TCP are contaminated with the toxic dioxin isomer 2,3,7,8,-tetrachlorodibenzo-*p*-dioxin (TCDD) which has been found to be carcinogenic in laboratory animals.[7]

---

[1] Hardell L, Sandström A. Case-control study: soft tissue sarcomas and exposure to phenoxyacetic acids or chlorophenols. *Br J Cancer* 1979; **39**: 711–17.

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Zack and Suskind[3] describe a male with chloracne after exposure in a TCP process accident at Monsanto Co. in 1949. He died in 1978 at age 58 with malignant fibrous histiocytoma of soft tissue origin.[3] (In the paper by Zack and Suskind this case is coded as ICD 173·9; however, when the diagnosis was presented to our consulting nosologist, ICD 171 was the code of choice.) Cook et al.[4] reported a male who worked on TCP production at Dow Chemical U.S.A. and had facial dermatitis; no diagnosis of chloracne was made. He died of fibrosarcoma in 1975 at age 53. Case 3 (J. H. Zack,[5] unpublished) worked on 2,4,5-T synthesis at Monsanto Co. and died in 1972 at age 49 of a generalised liposarcoma. No history of chloracne was reported. Only one cohort, reported by Ott et al. of 2,4,5-T workers at Dow, contained no case of soft tissue sarcoma.[5]

None of the four cohorts individually resulted in a reported excess risk for soft tissue sarcoma. When combined, however, the three cases from the four cohorts suggest a common pattern. This finding supports the concept of combining small cohorts of workers with a common occupational exposure for analyses. Such an effort to assemble an exposure registry of all people in the U.S. who have worked at the synthesis of 2,4,5-T is underway at the National Institute for Occupational Safety and Health (N.I.O.S.H.) the mortality experience of the combined cohort will be analysed retrospectively and followed up prospectively. This approach will provide greater statistical power than would be available for any subgroup of the registry analysed alone. N.I.O.S.H. has also embarked on a case-control study for soft tissue sarcoma to review occupational risk factors such as exposure to the phenoxy herbicides and chlorophenols, and to test further the results obtained in the Swedish case-control studies.

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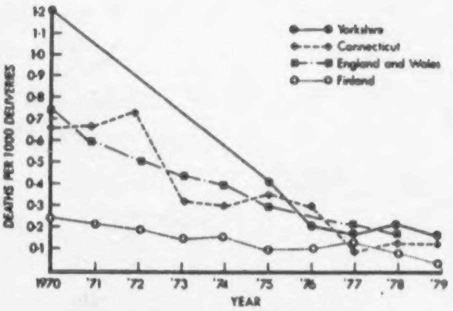
W. E. HALPERIN

## ANTENATAL ANTI-D PROPHYLAXIS

SIR,—Dr Bowman (Dec. 20/27) claims that the incidence and severity of rhesus haemolytic disease of the newborn (Rh HDN) is less in Britain than it is in certain parts of Canada and Sweden. Published incidence figures do not support his claim (table). It is very difficult to compare incidence data for Rh HDN for different areas of the world. One problem is that some reports are of "clinical" HDN and others "serological" HDN. If a serological test is done around the time of delivery and it is of the "enzyme only" type, the infant's direct antiglobulin test will be negative and so the case may not be included in the statistics. When antibodies are detected in the next pregnancy it will be labelled as a "new" case. So the incidence of HDN will very much depend on the timing of the test and the serological techniques used.

This, I believe, is one of the main reasons for the apparent disparity in the reported incidence of Rh antibodies developing during pregnancy. For primigravidae, as Woodrow[1] has pointed out, the reported incidence varies between 0·1% and 5·6%; in Leeds it is around 0·9%. As Bowman[2] points out in the Winnipeg series, if they had not used an enzyme autoanalyser technique, 29 of the 62 cases of sensitisation during pregnancy would not have been detected even 3 days after delivery. Another reason why their results are not strictly comparable is that they include the first 3 days after delivery in their "antenatal" incidence whereas our figures are of antibodies detected up to the time of delivery. Thus in figures cited by Dr Zipursky at the McMaster conference, although Winnipeg had an apparent high incidence of Rh immunisation during first pregnancies in comparison with other centres, they were unique in having no mothers out of a total of 3533 who developed antibodies in the first 6 months post-partum if anti-D immunoglobulin was given. The success rate, or otherwise, of the various procedures to reduce the incidence of Rh antibodies is perhaps better reflected in the stillbirth and neonatal death rates due to Rh shown in the figure.



Stillbirths and neonatal deaths due to anti-D sensitisation.

Bowman states that it is his experience that anti-D Ig does not modify the future serological response in mothers bearing Rh positive infants as we have claimed.[3] The problem here is that again we are not necessarily comparing like with like. We divided the patients into groups, and groups 1 and 2 consisted of cases where the anti-D Ig had been given when no antibodies were detectable in the mothers' serum at delivery, but the group 3 cases had detectable antibodies of the "enzyme only" type at delivery when the anti-D Ig globulin was given. Although one mother in group 3 developed a strong antibody in the next Rh positive pregnancy, all the babies were mildly affected. We have collected further data on this important aspect of Rh prevention and plan to publish them. We found that if anti-D Ig is given to a mother when no antibodies are detectable in her serum, her prognosis for further Rh positive infants is good, and if antibodies are detectable in a mother's serum at delivery, by both the enzyme and IAG techniques, then anti-D Ig usually does not modify the future immunological response.

Our study of deaths due to Rh have highlighted the importance of the mother who develops antibodies in her first Rh positive pregnancy. There is a 30% chance that, if the next infant is Rh D positive, it will die, and because of this we are strongly in favour of

CASES OF RHESUS (ANTI-D) SENSITISATION PER 1000 BIRTHS

| Area | 1964 | 1967 | 1968 | 1969 | 1970 | 1971 | 1972 | 1973 | 1974 | 1975 | 1976 | 1977 | 1978 | 1979 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Yorkshire, U.K. |  | 10 |  |  | 8·6 | 7·9 | 6·9 | 6·2 | 4·75 | 4·4 | 3·6 | 3·6 | 3·6 | 3·2 |
| Manitoba, Canada | 10·6 |  |  |  |  |  |  |  |  | 3·5 | 2·7 | 2·0 |  |  |
| Connecticut, U.S.A. |  |  |  |  | 6·3 | 5·2 | 4·6 | 3·1 | 2·6 | 2·5 | 1·5 | 2·1 | 1·5 | 1·6 |
| Finland |  |  |  |  | 3·2 | 2·8 | 2·4 | 2·25 | 2·6 | 2·2 | 1·8 | 1·8 | 2·2 | 1·3 |
| U.S.A. |  |  |  |  | 4·5 | 4·0 | 3·71 | 2·74 | 2·27 | 2·06 |  |  |  |  |
| Western Australia |  |  |  |  |  |  |  |  | 1·17 | 1·5 | 1·4 |  |  |  |
| Lanarkshire, U.K. | 9 |  |  |  |  |  |  |  |  |  |  |  |  | 3·8 |

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STATEMENT OF
HARRY N. WALTERS
ADMINISTRATOR OF VETERANS AFFAIRS
BEFORE THE
SUBCOMMITTEE ON COMPENSATION, PENSION AND INSURANCE
COMMITTEE ON VETERANS AFFAIRS
HOUSE OF REPRESENTATIVES
APRIL 26, 1983

MR. CHAIRMAN AND MEMBERS OF THE SUBCOMMITTEE:

I AM PLEASED TO BE HERE TODAY TO STATE THE VETERANS ADMINISTRATION'S POLICY CONCERNING THE COMPLEX, FAR-REACHING ISSUES RAISED BY THE USE OF AGENT ORANGE IN VIETNAM. AS YOU KNOW, THE CHIEF BENEFITS DIRECTOR, THE CHIEF MEDICAL DIRECTOR, AND OTHER MAJOR REPRESENTATIVES OF THE VETERANS ADMINISTRATION WILL, PURSUANT TO THE COMMITTEE'S REQUEST, PRESENT TESTIMONY AS TO THE SPECIFIC ISSUES TOMORROW. I WOULD ALSO LIKE TO EXPRESS MY APPRECIATION TO MR. CURRIEO, THE COMMANDER-IN-CHIEF OF THE VETERANS OF FOREIGN WARS, FOR EXTENDING TO ME THE COURTESY OF YIELDING A FEW MINUTES OF HIS TIME SO THAT I MAY MAKE THE FOLLOWING STATEMENT. AS PER OUR AGREEMENT, MR. CHAIRMAN, TO ENABLE MR. CURRIEO TO DEPART HERE ON SCHEDULE, I WILL BE PLEASED TO RESPOND TO ANY QUESTIONS SUBMITTED TO ME FOR THE RECORD RATHER THAN HERE TODAY.

THE USE OF AGENT ORANGE IN VIETNAM HAS CAUSED MUCH CONCERN AND GIVEN RISE TO CONTROVERSY. ALTHOUGH WE ARE HERE TODAY TO DISCUSS A PROPOSAL OF PRESUMPTIVE LAW FOR PURPOSES OF COMPENSATION, THE ISSUES OF HERBICIDE EXPOSURE AND ITS POSSIBLE EFFECTS ARE, IN FACT, OF MUCH

BROADER, WORLDWIDE CONCERN. THE OVERRIDING ISSUE IS WHETHER HERBICIDE EXPOSURE HAS LONG-TERM EFFECTS ON THE HEALTH OF NOT ONLY VIETNAM VETERANS BUT OF ALL HUMANS.

AT MY CONFIRMATION HEARING, I PLEDGED THAT I WOULD BE THE ADVOCATE FOR AMERICA'S VETERANS. I PROMISED TO ADMINISTER THE LAWS ENACTED ON THEIR BEHALF FAIRLY, EFFECTIVELY, AND COMPASSIONATELY. I HAVE AND WILL CONTINUE TO DO ALL THAT I CAN TO INSURE THAT VIETNAM VETERANS WHO MAY HAVE BEEN EXPOSED TO PHENOXY HERBICIDES RECEIVE HEALTH CARE FOR DISABILITIES THAT COULD POSSIBLY BE ASSOCIATED WITH SUCH EXPOSURE IN ACCORDANCE WITH THE PROVISIONS OF PUBLIC LAW 97-72. I VIEW MY RESPONSIBILITIES AND OBLIGATIONS AS A SACRED TRUST.

I FULLY APPRECIATE THE TIME IT IS TAKING TO PERFORM THE MEDICAL RESEARCH AND OBTAIN THE MEDICAL EVIDENCE ESSENTIAL TO A RESOLUTION OF THESE CRITICAL ISSUES. I CAN ASSURE YOU THAT I AM COMMITTED TO ACTIVELY, FULLY, AND ENTHUSIASTICALLY SUPPORTING THE NUMEROUS MEDICAL STUDIES NOW UNDERWAY, OR PLANNED, WHICH WILL DETERMINE WHETHER EXPOSURE HAS LONG-TERM HEALTH CONSEQUENCES.

SINCE JANUARY 1ST OF THIS YEAR, THE VETERANS ADMINISTRATION HAS, AMONG OTHER THINGS: UNDERTAKEN TO IMPROVE ITS AGENT ORANGE REGISTRY. WE HAVE EXPANDED ITS EFFORTS TO PROVIDE INFORMATION CONCERNING AGENT ORANGE TO VETERANS AND OTHER CONCERNED PARTIES. WE HAVE TRANSFERRED, BY INTERAGENCY AGREEMENT, THE PERFORMANCE OF THE EPIDEMIOLOGICAL STUDY AND THE SUPPORTING RESOURCES TO THE CENTER FOR DISEASE CONTROL. WE HAVE REORGANIZED AND REVITALIZED THE CHLORACNE TASK FORCE. WE HAVE IMPROVED THE COMMUNICATIONS AND

COORDINATION BETWEEN KEY OFFICIALS IN THE VA, MOST NOTABLY OUR AGENT ORANGE PROJECTS OFFICE, AND OUR FIELD PERSONNEL.

IN ADDITION, THE FOLLOWING STUDIES ARE UNDERWAY:

1) A VIETNAM VETERANS IDENTICAL TWIN STUDY
2) VIETNAM VETERANS MORTALITY STUDY
3) WITH THE ENVIRONMENTAL PROTECTION AGENCY, A RETROSPECTIVE DIOXIN/FURAN ADIPOSE TISSUE STUDY
4) UPDATE OF THE ANALYSIS OF THE WORLDWIDE LITERATURE ON HERBICIDES
5) SPONSORING THE AUTHORSHIP OF A MONOGRAPH SERIES BY INTERNATIONALLY RECOGNIZED EXPERTS
6) CONTINUING SOLICIATIONS OF ADDITIONAL SPECIAL RESEARCH PROJECTS FROM VA SCIENTISTS.

THE AIR FORCE IS COMPLETING THE RANCHHAND STUDY, THE RESULTS OF WHICH SHOULD BE AVAILABLE IN JUNE OF THIS YEAR. FURTHER MEDICAL RESEARCH AND OTHER STUDIES REGARDING THE POSSIBLE HEALTH EFFECTS OF HERBICIDE EXPOSURE HAVE BEEN AND ARE BEING CONDUCTED BOTH WITHIN THE UNITED STATES AND AROUND THE WORLD. THE VA HAS RESPONDED TO THE CONTROVERSIES RAISED BY THE SWEDISH PAPERS SUGGESTING DIOXIN AS A POSSIBLE CAUSATIVE FACTOR OF SOFT TISSUE SARCOMA.

The Veterans Administration with the Armed Forces Institute of Pathology, has begun an epidemiological study of soft tissue sarcoma. The Center for Disease Control has announced its intention to conduct a similar epidemiological study. Public health authorities in the States of New York, Washington, and Kansas have likewise announced studies of soft tissue sarcoma.

The endeavors of the Veterans Administration and others to obtain the medical evidence necessary to resolve these complex issues is now broad-based and on-going. It is my hope that the discussions which follow today and in the months ahead focus on the issues. The VA is now attempting to search for the final medical conclusions to the complex Agent Orange exposure issue. It is my desire that this agency stand as a beacon of hope in its search for the final and conclusive answers and not as a whipping post for the debate. The medical community has reached a consensus that a causal relationship exists between exposure to dioxins and chloracne, a skin disorder that appears within a relatively short period after exposure. The medical community has also reached a consensus that, in some individuals, porphyria cutanea tarda (PCT), a liver disorder, can be triggered by dioxin exposure. However, no medical studies have been concluded which establish a causal connection between exposure and the initial occurrence of these disabilities long after exposure. Accordingly, the Veterans Administration has recognized both chloracne and PCT as compensable disabilities attributtable to service connection.

THERE IS PRESENTLY NOT A CONSENSUS WITHIN THE MEDICAL COMMUNITY THAT A CAUSAL CONNECTION BETWEEN PHENOXY HERBICIDE EXPOSURE AND LONG TERM HEALTH CONSEQUENCES IN HUMANS EXISTS. THERE ARE SEVERAL STUDIES WHICH, ON A CORRELATIVE BASIS, SUGGEST THAT EXPOSURE TO DIOXIN AND/OR PHENOXY HERBICIDES MAY BE RELATED TO SOFT TISSUE SARCOMAS. THERE ARE MANY OTHER STUDIES THAT DO NOT SUBSTANTIATE THIS SUGGESTED RELATIONSHI~~O~~P. TO DATE, THERE ARE ONLY THEORIES AND HYPOTHESES.

H.R. 1961 IS BASED ON THESE THEORIES AND HYPOTHESES. IT ENDEAVORS TO PRESUME THAT WHICH IS NOT, AS YET, MEDICALLY ACCEPTED OR PROVEN. H,R. 1961 IS NOT BASED ON EVIDENCE ACCEPTED BY THE MEDICAL COMMUNITY AS RELIABLE; IT IS NOT BASED ON FACT. GIVEN THE FAR-REACHING CONSEQUENCES OF THE PUBLIC POLICY ADDRESSED IN PART IN H.R. 1961, I DO NOT BELIEVE THAT CONGRESS SHOULD BASE ITS DECISIONS ON HYPOTHESES.

I, AS ADMINISTRATOR OF VETERANS AFFAIRS, HAVE AN OBLIGATION TO SAFEGUARD THIS NATION'S COMPENSATION PROGRAM FOR SERVICE-DISABLED VETERANS AND THE SURVIVORS OF THOSE WHO GAVE THEIR LIVES FOR THEIR COUNTRY. THE PURPOSE OF THIS PROGRAM IS TO COMPENSATE INDIVIDUALS FOR INJURIES OR DISEASES CONTRACTED IN OR AGGRAVATED BY MILITARY SERIVCE. THE EXISTENCE OF A CAUSAL CONNECTION BETWEEN DISABILITIES AND MILITARY SERVICE TO THE NATION IS THE FUNDAMENTAL PRECEPT OF THE COMPENSATION SYSTEM. IT IS THE BASIS OF ITS INTEGRITY AND ITS ACCEPTABILITY. SHOULD H.R. 1961 BECOME LAW, THE BASIC PREMISE OF THE PROGRAM WOULD BE FUNDAMENTALLY CHANGED AND ITS CONTINUED VIABILITY JEOPARDIZED.

I MUST ALSO EXPRESS MY CONCERN WITH H.R. 1961'S POTENTIAL FOR ALARMING MILLIONS OF VETERANS AND THE POPULATION AS A WHOLE. MOST PEOPLE WILL NOT UNDERSTAND THE CONCEPT OF PRESUMPTION AS EXPRESSED IN H.R. 1961, I.E., NOT BASED ON ACCEPTANCE BY THE MEDICAL COMMUNITY OF A CONNECTION BETWEEN EXPOSURE TO HERBICIDES AND THE OCCURRENCE OF SERIOUS ILLNESSES LONG AFTER EXPOSURE. RATHER, THERE IS A SERIOUS RISK THAT THE MESSAGE CONVEYED WILL BE THAT HERBICIDE EXPOSURE HAS IN FACT BEEN MEDICALLY ACCEPTED AS CAUSING, AMONG OTHER THINGS, CANCER. WE MUST REMEMBER THAT HERBICIDE EXPOSURE IS OF A WORLD-WIDE CONCERN.

AS ADMINISTRATOR OF VETERANS AFFAIRS, I AM COMMITTED TO THE RAPID RESOLUTION OF THE AGENT ORANGE ISSUE. IF A CONCENSUS OF THE MEDICAL COMMUNITY FINDS WITH REASONABLE MEDICAL CERTAINTY THAT EXPOSURE CAUSES DISABILITIES, I WILL INSURE THAT OUR VETERANS ARE FAIRLY COMEPNSATED FOR THESE DISABILITIES. THE VETERANS ADMINISTRATION WILL, OF COURSE, CONTINUE TO PROVIDE HEALTH CARE TO THOSE VETERANS WHO MAY HAVE BEEN EXPOSED TO PHENOXY HERBICIDES FOR DISABILITIES THAT COULD POSSIBLY BE ASSOCIATED WITH SUCH EXPOSURE.

THANK YOU MR. CHAIRMAN AND MEMBERS OF THE SUBCOMMITTEE.

VA Responses to Questions Submitted by Rep. Mica in Connection with April 27, 1983 Hearing on H.R. 1961, 98th Congress.

Question: 1. In a recent article in the Washington Post, VA Administrator Harold Walters was quoted as stating that Vietnam veterans will not have to wait for scientists to prove a direct link between the herbicide Agent Orange and certain kinds of ailments to qualify for disability benefits. The Administrator went on to say that if the government's epidemiological study shows that Vietnam veterans, as a group, are suffering a "statistically significant" rate of health problems that is higher than the rate for non-veterans, the VA will consider those ailments service-connected. Therefore, it is my understanding from these statements that if the epidemiological study comes out positive for certain diseases that it could serve as a basis for compensation. I would like to know specifically if the CDC epidemiological study is going to serve as basis for policy for VA compensation for Agent Orange?

Answer: We will carefully review each report based on the CDC epidemiological study as well as other studies that may be completed before the CDC study. If a statistical correlation between Agent Orange exposure and a specific disease or condition is shown, we have the authority to amend our regulations to authorize service connection for the disease or condition. Because we cannot predict the study findings, we cannot at this time determine whether the CDC study, or any other study, will afford a basis for amendments to our regulations.

Question: 2. What is the anticipated cost of the epidemiological study and is the VA going to reauthorize funding for this study every year?

Answer: The cost for the total epidemiological study is tentatively estimated at as much as $100 million. The Centers for Disease Control will submit detailed budget requests annually for inclusion in the VA's budget, which is submitted to the Congress for its approval.

Question: 3. It is general principle in the field of epidemiological studies that rarer conditions can only be determined in large populations. Is the sample size of the CDC epidemiological study large enough to pick up rare conditions?

Answer: Since the Centers for Disease Control (CDC) is in the process of developing the protocol for the epidemiology study, it is difficult for us to comment on its study. In general, however, the sample size for a cohort study such as the epidemiology study depends on many factors.

1) the frequency of disease incidence or mortality rates in the reference population

2) minimum value for the relative risks which is desired to detect

3) the desired level of statistical significance and

4) the desired study statistical power.

Obviously, one will need a larger sample and longer follow-up to detect a small increase over background rates. In addition, in most cases, detection of relatively rare conditions, such as soft-tissue sarcoma requires a large sample size. On the other hand, if the risk of developing a specific health outcome is much greater than that expected in the general population, the sample can be smaller. We believe another epidemiology methodology, the case-control study is better suited to the study of rare diseases or those with long latency periods. It is our understanding that CDC is proposing just such a study for soft-tissue sarcoma.

Question: 4. The VA has been monitoring medical treatment being given to veterans claiming ailments relating to Agent Orange as directed under Pub. L. No. 97-72. In your views are veterans receiving the level of attention laid out in the law?

Answer: Following enactment of Pub. L. No. 97-72, the VA issued interim guidelines for the implementation of this legislation. Public comments were solicited prior to the promulgation of final guidelines in September 1982. A brochure describing the eligibility criteria for care and treatment under the authority of this law was prepared and widely disseminated. A tracking mechanism to collect data on veterans receiving care under Pub. L. No. 97-72 was established effective July 1, 1982. From February 1982 to February 1983, about 9,400 Vietnam veterans were admitted for inpatient care related to Agent Orange and there were approximately 369,000 outpatient visits by Vietnam veterans utilizing their eligibility under Pub. L. No. 97-72. We believe that this demonstrates that veterans are availing themselves of this eligibility and we believe that they are receiving the medical care and treatment they deserve.

Medical Centers and independent outpatient clinics are continuing to collect data on veterans reporting for medical care or treatment under Pub. L. No. 97-72. On September 15, 1982, final guidelines regarding health care services offered under this law were issued. The guidelines were published in the Federal Register on November 17, 1982. Information about the services available under this legislation has been widely disseminated through the VA Vet Center Program, a new series of pamphlets, and the assistance of veterans' service organizations. While the tracking mechanism is in the process of fine-tuning, preliminary data indicate that veterans have been seen in great numbers under the provisions of this law, and that they are receiving proper attention.

Question: 5. The GAO in its October 1982 report on the Agent Orange Examining Program, criticized the VA's Agent Orange Registry for a number of inadequacies and recommended that the registry be discontinued. What is the status of the registry today?

Answer: On March 1, 1983, DM&S Circular 10-83-38, entitled "Possible Exposure of Veterans to Herbicides During the Vietnam War" was issued to all VA health care facilities. This circular provides for streamlining of Agent Orange Registry procedures and features an improved registry code sheet which will serve to improve the registry data gathering process. The new revised registry code sheet will obtain the veteran's address, sex, specific diagnosis for the veteran's health problems and other related information. This information was not obtained in a computerizable form previously and there is a demonstrable need to have this information readily available. The registry will be reviewed periodically for quality assurance.

Question: 6. I would like to know how applicable the findings of the Ranch Hand study are to Agent Orange victims as a whole? I believe the Ranch Hand participants had showers following their exposure to Agent Orange, whereas ground troops were unaware of their exposure while it was sprayed from planes above them. In addition it is my understanding that the participants in the Ranch Hand Study have been found to be healthier than the general population. Would this factor influence the results of this study?

Answer: Many of the civilian studies reported in the scientific literature were limited by problems of methodology, small study group size, and/or inadequate time intervals to assess truly long-term effects from exposures to dioxin (TCDD). The Alsea Oregon study has been sharply criticized for deficiencies in data-gathering methods, and statistical analysis. Only six years have elapsed since the industrial accident in Seveso, Italy, and despite the large size of the study population, sufficient time has not elapsed to form conclusions as to the presence or absence of long-term effects of health. Most studies of groups exposed to phenolic com[illegible] and TCDD in an industrial setting have i[illegible]ps of fewer than 300 exposed individuals, [illegible]d th[illegible] small numbers severely limit the abili[illegible]atistically-valid conclusions for or [illegible] th effects arising from these exposures. The Air Force Health Study has attempted to overcome these limiting factors by submitting the study design to an extensive peer review process, by selecting a population for study which had exposure 11-20 years ago, and by attempting to maximize the group size (approximately 1,200). This approach should make the Air Force Health Study as or more valid than previously-conducted studies of civilian populations.

The Air Force has published the Study Protocol for the Air Force Health Study. The protocol is available as Air Force Technical Report SAM-TR-82-44 "Epidemiologic Investigation of Health Effects in Air Force Personnel Following exposure to Herbicides: Study Protocol"; NTIS ID No. A 122250. It was released December 1982 by the USAF School of Aerospace Medicine, Brooks AFB, Texas 78235. The following data on exposure is taken directly from the Protocol (pg 50-51):

### "Exposure Concepts"

"A major concern in conducting this study is the lack of accurate exposure data. Although most personnel assigned to RANCH HAND squadrons were undoubtedly exposed to Herbicide Orange and TCDD, the exposures within the group must have varied widely. Exposure to herbicides and TCDD by RANCH HAND personnel occurred almost daily. Anecdotal information suggests that many had direct skin contact which was repetitive over a long period of time (one-year tour for most individuals). Further, it is also suggested that most RANCH HAND personnel felt that the herbicides employed in the operations were not toxic to animals and man, and hence, they did not exercise the caution in handling these chemicals that is recommended today.

"From a historical review of RANCH HAND operations, it appears most individuals can be classified into one of three groups based on their likely potential for exposure to the herbicides:

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| (1) | Pilots, Co-pilots and Navigators: | low potential |
| (2) | Crew Chiefs, Aircraft Mechanic, and other Support Personnel: | Moderate potential |
| (3) | Console Operators and Flight Engineers: | high potential |

"The 'pilot' group received most of their exposure during pre-flight checks as well as during the actual dissemination missions. The crew chief group; experienced contact with herbicides during dedrumming and aircraft loading operations, as well as during on-site repair of the aircraft and spray equipment. The console operator group was exposed while supervising the loading of the aircraft, during ground testing of eqipment, and by tank leakage during dissemination missions.

"The available historical records on Operation RANCH HAND indicate that personnel assigned to the project seldom had a 'routine' work schedule or environment, thus complicating estimates of the level of herbicide and dioxin exposure. Since actual exposure data (e.g., mg of herbicide/kg body wt) are not available, an exposure index will be used. The exposure indices will be calculated for each RANCH HAND individual to obtain frequency distribution, and will be calculated by evaluating the known factors that would have influenced exposure. These will include such factors as:

(1) Date of tour with RANCH HAND in Vietnam.
(2) Number and lengths of tours in Vietnam with RANCH HAND.
(3) Number of herbicide dissemination missions (as reflected by flying hours and air medals).
(4) Herbicides employed (records are available that reflect the amount of each herbicide sprayed each month and year).
(5) Crew position.
(6) Routes of exposure (the major route of exposure for most RANCH HAND personnel was probably percutaneous, although exposure through inhalation may have also been significant)."

Last fall (1982), the Air Force released its preliminary finding on mortality. It noted:

> "Although more extensive analyses and comparisons remain to be done, preliminary findings indicate that the overall crude mortality of the RANCH HAND and comparison groups have been very similar. Based on the 60 deaths identified, excluding the 22 killed in action no statistically significant differneces in total death rates have been found between the RANCH HAND group and the comparison group. Both groups appear to have experienced significantly less mortality than a similarly aged U.S. white male population, indicating a healthy worker effect. However, thus far, very few deaths have occurred in the study groups, and these deaths represent only a very early assessment of mortality. The only preliminary interpretation that can be made from these data is that, thus far, the RANCH HAND group has had a mortality experience equivalent to that of an occupationally similar comparison group. Periodic reassesments of the mortality experience of the groups will be made. Definitive conclusions must await the completion of more detailed analyses and the accumulation of a larger number of deaths in the study groups in the coming years."

The fact that the RANCH HAND population and its matched control show a healthy worker effect would be consistent with data showing that healthy men are selected for military duty, as well as the availability of health care throughout their careers and retirement.

Question: 7. As you know, there is a Swedish study which links soft tissue sarcoma to exposure to phenoxy herbicides. It is my understanding that the VA contends that the results of the Swedish study are not enough to show a connection. What I would like to know is if this study is not enough, does it mean our epidemiological study is not going to be enough to determine a connection between Agent Orange and certain diseases?

Answer: The epidemiological study mandated by Pub. L. No. 96-151, as amended by Pub. L. No. 97-72, should provide the answer to the question of the relation between exposure to Agent Orange in Vietnam and the subsequent appearance of many diseases. There are certain diseases, however, that occur so infrequently in the general population that they may escape detection in an epidemiological study using an approach known as a cohort study. In those instances, a case-control study should provide suggestive findings of an etiological relationship between exposure and the subsequent development of disease. (See our response to question 3.) It is also possible that some cancers may develop so long after exposure that they may escape detection unless the participants in the epidemiological study are followed for several decades.

Question: 8. How much scientific evidence is necessary to make veterans eligible for Agent Orange compensation?

Answer: If scientifically valid studies enable the medical community to reach a consensus that certain diseases or conditions result from Agent Orange exposure, the VA has the authority to award compensation to veterans in appropriate cases. "Scientific validity" assumes supportable, verifiable conclusions based on sound methodology with full disclosure of all data.

Question: 9. We are all aware of the recent award to the citizens of Times Beach, Missouri, for their exposure to dioxin. I would like to know the difference between exposure to dioxin in Times Beach and the exposure of our troops in Southeast Asia? How can we compensate in one case and not in the other?

Answer: Residents of Times Beach have *not* been promised, nor have they received, any compensation for having been exposed to dioxin. Rather, they have received relocation assistance that includes purchase of their homes. This was done in view of the *potential* hazard to families living where soils are known to be contaminated with relatively high levels of dioxin. This Federal Government assistance is somewhat similar to disaster assistance and *not* comparable to VA compensation, which by statute can be paid only for actual disability within the meaning of section 101(16) of title 38, United States Code.

Question: 10. Has the VA or Department of Defense have any way of determining what areas in Southeast Asia were sprayed with phenoxy herbicides and what troops were directly sprayed with it? Have they any numbers of how many American civilians working in Southeast Asia and the U.S. during the Vietnam era were exposed to these herbicides?

Answer: The Veterans Administration, the Department of Defense, and the Agent Orange Working Group's Scientific Panel have spent many hours with the personnel of the Army Agent Orange Task Force in order to develop methods for defining and determining the extent of exposure of ground troops to Agent Orange in Vietnam. It is now thought that a satisfactory, if not completely accurate, separation can be made between veterans who were likely to have been heavily exposed and those who were unlikely to have had contact with the herbicide. Groups with an intermediate likelihood can also probably be identified.

The available information on the use of phenoxy herbicides and troop distribution in Vietnam is maintained by the Department of Defense. We have no information on whether that Agency maintains records of civilians.

VA responses to questions submitted by Rep. Applegate in connection with April 27, 1983 hearing on H.R. 1961, 98th Congress.

Question: 1. A witness commented on the quick action of the Administration in the Times Beach buy-out versus the lack of action on the issue of compensation for Vietnam veterans exposed to Agent Orange. Please furnish your response to the comment by this witness.

Answer: The situations in Vietnam and in Times Beach are very dissimilar, notwithstanding that dioxin is involved in both instances. On the average, in Times Beach, dioxin was present in the soil in concentrations in the range of 300 parts per billion; in Vietnam the soil concentration of dioxins would have amounted to less than 0.02 parts per billion even if Agent Orange was sprayed directly on the soil rather than onto dense jungle vegetation.

In Times Beach, a decision was made to remove a continuing potential hazard by buying contaminated property. Individuals living in the Times Beach area were potentially exposed to dioxin over a prolonged period of time and children played in the contaminated soil. Failure to take action now could result in continuing exposure into the future. The action taken was a preventive one to reduce the risk of possible future harm by eliminating the exposure. No person in Times Beach was compensated for adverse health effects due to dioxin but rather for the loss of property value.

In the matter of Vietnam veterans, there is no potential danger from continuing exposure. Exposure of the veterans to Agent Orange ended ten or more years ago. There is no clear evidence that that exposure resulted in harm or injury that would persist or first become apparent many years after the last possible exposure.

Action taken to reduce the risk of possible future harm can be based on decidedly different information than action to compensate individuals for actual harm which arose in the past. That distinction explains, what on the surface, appears to be inconsistent actions.

Question: 2. In H.R. 1961, it is said that Agent Orange exposure "may" cause certain disorders and veterans "shall" be compensated. Is there any precedent for this in title 38, United States Code?

Answer: No.

Question: 3. You say your current authorities are adequate regarding chloracne and PCT. Please explain to the Members what you mean.

Answer: The policy of the VA is to grant service connection for any condition attributable to military service (other than a condition resulting from the veteran's willful misconduct), no matter how long after service the condition became manifest. When medical evidence establishes a lengthy latent period or previously unrecognized cause-and-effect relationship, our authority under sections 210(c)(1) and 355 of title 38, United States Code, is ample to permit promulgation of rules to assure proper consideration of claims and adequate compensation to afflicted veterans.

Question: 4. The statement was made that VA is insisting on evidence far beyond that which would be required "in a court of law." Please respond.

Answer: In civil litigation generally, the plaintiff must establish his or her case by a preponderance of the evidence. As a Federal agency, we must assure that our conclusions rest on a rational basis. The contention to which you allude implies that a "preponderance of the evidence" has already established certain disorders as long-term effects of dioxin exposure. This is not the case. There is no evidence suggesting that dioxin exposure results in the delayed development of chloracne or porphyria cutanea tarda. There is some evidence, not by any means a preponderance, suggesting that phenoxy herbicide exposure may result in the delayed development of soft-tissue sarcomas; this evidence (the Swedish studies discussed in our testimony on H.R. 1961) is countered by other evidence tending to negate any such cause-and-effect relationship. We are satisfied that our policy determination respecting these matters is sound.

Question: 5. Please explain the adjudicative process, particularly with regard to burden of proof and VA's ex parte approach.

Answer: Our purpose in claims adjudication is to grant each claimant every benefit that can be supported in law. The process is informal and non-adversarial. After the claim is filed, VA assists the claimant in developing the facts pertinent to the claim. This includes obtaining the records of military service and medical treatment basic to most claims. If the records and other evidence are insufficient, the claimant is clearly advised as to what further evidence is needed and what sources are acceptable. The claimant may request a hearing on any issue and may present witnesses and argument. All evidence and argument is included in the record, and the entire record is considered without regard to formal evidentiary rules. If an adverse determination is made, the claimant is advised of appellate rights. If a claimant pursues an appeal, the Board of Veterans Appeals conducts a complete and independent review of the claim, on the basis of the evidence of record, which may include a hearing before the Board. The Board may request the development of further evidence, or, in cases of unusual medical complexity, an independent medical expert's opinion.

In view of our active participation in assisting in the development of claims, we do not employ the concept "burden of proof." However, pursuant to section 3.102 of title 38, Code of Federal Regulations, the claimant's evidence must be sufficient to justify a belief in a fair and impartial mind that the claim is well grounded. That section also sets forth the "reasonable doubt" rule, which applies after all the evidence is assembled and the evidence against the claimant on any material issue is in approximate balance with the evidence favoring the claimant on that point. When this occurs, the "reasonable doubt" rule provides for the issue to be resolved in the claimant's favor.

Question: 6. Do your regulations require that a causal relationship be shown between disability and something that happened in service?

Answer: Our regulations carry out the statutory concept that "service connection" implies a causal relationship between something that happened in service and subsequent disability.

With respect to disability, the term "service-connected" is defined in section 101(16) of title 38, United States Code, as disability incurred or aggravated in line of duty during active military, air, or naval service. Hence, our regulations provide that service connection may be established by evidence of incurrence or aggravation (of a pre-existing disability) in service, or by the application of sound medical principles relating the disability to the period of service. Under certain circumstances, service connection can also be established by the application of the statutory presumptions found in section 312 of title 38, United States Code.

Question: 7. If your adjudicating agency encounters evidence of a skin condition or a liver condition in service and there are ratable residuals after service, do you grant service connection or inquire into etiology of the disability and whether it was caused by service?

Answer: In the hypothetical you pose--evidence in the service records of a skin or liver condition plus ratable residuals of that condition after service-- there would be no need to inquire into etiology. Service connection is in order for any disability not the result of the veteran's willful misconduct, incurred or aggravated during military service. It would not be necessary to establish that the disability resulted directly from the performance of military duty.

Question: 8. Regarding presumptive service connection, doesn't the Administrator have authority to add to the list of disabilities in title 38, U.S. Code 312? So really what H.R. 1961 would give you would be the right to add to the list without the one year time limitation and presumably limited to the same universe benefited by H.R. 1961. Is that right?

Answer: Yes, we do have authority, pursuant to section 301(3) of title 38, to add to the list of chronic diseases for which the section 312(a)(1) presumption is available. The authority that would be granted by H.R. 1961 incorporates a standard ("diseases... that medical research has shown may be due to exposure to herbicides, chemicals, medications, or environmental hazards or conditions") that, in our view, is unnecessary. As explained in our response to Question 3, we have ample authority, without the aid of a presumption, to grant service connection for a disability attributable to military service when medical evidence establishes a longer incubation period or a previously unrecognized cause-and-effect relationship.

Question: 9. Tell us about your rule relating to heart disease and amputations. Is it comparable to the presumptions proposed in H.R. 1961.?

Answer: Pub. Law No. 94-433 ordered a scientific study to determine whether there is a relationship between the amputation of an extremity and the subsequent development of cardiovascular disease. An epidemiological study was made by the Medical Follow-up Agency of the National Academy of Sciences. The resulting report analyzed statistically valid samples of veterans having service-connected extremity amputations matched by age and war period with nonamputee veterans. The report concluded that veterans who suffered an amputation of both legs or of one leg at or above the knee have a significantly higher risk of dying of diseases of the cardiovascular system.

In response to this report our regulations were amended to provide service connection for ischemic heart disease or other cardiovascular diseases developing in veterans with service connected amputations at the cited levels. Service connection is granted for cardiovascular diseases in amputees on the basis of the relationship established by the study, not by means of a presumption. This rule is not comparable to the statutory presumption that would be authorized by H.R. 1961.

Question: 10.A. (i) Please describe briefly the two major types of epidemiological studies.

Answer: Epidemiological research is generally divided between "case/control" and "cohort" studies. The two types differ as to how they are performed and what the results mean.

In a case/control study, the investigator identifies as "cases" individuals who have or have had a disease or, in the instance of soft tissue sarcoma, a group of questionably related diseases. Another set of individuals called "controls" is used for comparison. They are healthy or suffer from some other unrelated disease but who resemble the "cases" in other respects.

All living "cases" and "controls" are questioned about their exposure to the suspected cause of the disease being investigated. The nearest surviving relative of any deceased "case" or "control" usually is similarly questioned.

Any records available are examined for evidence to corroborate the results of questioning. These usually are employment records and rarely provide specific information regarding exposure to previously unrecognized hazards. In cancer investigations, both questioning and record examination are more difficult and less reliable because the suspected exposure occurs five to forty years before the research begins.

Case/control studies contain so many uncertainties that they are regarded as yielding suggestive findings at best. More definitive results must come from other types of investigation.

Cohort studies are usually more conclusive in establishing a cause-effect relationship between disease and exposure. Such a study begins with a group of people known to have been exposed to the suspected cause and called the "exposed" or "experimental cohort." It is compared to a "control cohort" resembling the exposed group as closely as possible but known not to have had contact with the suspected cause. Both cohorts are examined in the same way to determine how many members have the disease being investigated. The resulting numbers provide the comparison between cohorts and determine whether contact with the suspected cause has resulted in more disease among the exposed cohort. Cancer investigations are made more difficult by the long interval between the appearance of the disease and the exposure. Even with this limitation, cohort studies generally are considered more conclusive than case/control investigations.

Question: (ii) Which type were the Swedish studies?

Answer: The two Swedish investigations of soft-tissue sarcoma were both case/control studies.

Question: B. Do similar studies elsewhere in the world also uniformly incriminate dioxin in relation to soft tissue sarcoma?

Answer: No other systematic study of either the case/control or cohort type support the two Swedish studies, both of which were performed by the same group of investigators using the same methods and thus really two parts of one study.

Question: 11. Are the presumptions of causation that H.R. 1961 would create different from the presumptions now in title 38, U.S. Code § 312(a)?

Answer: Yes. The section 312(a) presumptions are not presumptions of causation. They are intended to assist veterans in establishing claims for compensation. The principal difference between the section 312(a) presumptions and those proposed by H.R. 1961 is that the former are time-specific, whereas the latter would authorize service connection no matter how long after service the veteran begins to experience symptoms. There are also other distinctions. The section 312(a) presumptions require 90 days of wartime service. The H.R. 1961 presumption does not require a minimum period of service. The section 312(a) presumptions benefit veterans of all war periods. The H.R. 1961 presumption is limited to veterans who served in Southeast Asia during the Vietnam conflict. Authority to grant service connection on the basis of the H.R. 1961 presumption would expire one year after submission to Congress of the first report on the Pub. L. No. 96-151 epidemiological study. Nothing in section 312(a) is comparable to this limitation, which arises out of the recognition that the epidemiological study, when completed, may not support the bill's hypotheses.

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# Review of Literature on Herbicides, Including Phenoxy Herbicides and Associated Dioxins

## Volume I

This report was prepared under a cost reimbursement-type contract awarded on a competitive bid basis on December 15, 1980. The contract will run for nine and one-half months and is funded at $111,743.

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TABLE 5-4: HEALTH EFFECTS FROM INDUSTRIAL ACCIDENTS INVOLVING TCDD

| Year | Location | No. Affected | Organ Systems Affected: |  |  |  |  |  |  |  |  |  |  |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
|  |  |  | Dermal | Renal | Hepatic | Neuro. | Asthenia | Blood | GI | Lipid Metab. | Immuno. | CV | Pulm. |
| 1949 | Nitro, W.Va. | 228 | + |  | + | + | + | + |  | + |  |  | + |
| 1953 | BASF, W. Ger. | 55 | + | + | + | + | + |  |  |  |  | + | + |
| 1956 | Grenoble, Fr. | 17 | + |  | + |  | + |  |  |  |  |  |  |
| 1963 | Amsterdam, Neth. | 44 | + |  | - |  | + |  | + |  |  |  |  |
| 1968 | Bolsover, Derbyshire U.K. | 79 | + |  | - |  | + | + | - |  |  |  |  |
| 1976 | Seveso, Italy | 134 | + |  | + | + | + | + | + |  | + | - |  |

Code: (+) An effect was observed;

(-) An effect was not observed in patients examined (or questioned) about the effect;

( ) Patients were not examined or questioned, regarding any effects in the indicated organ system.

Amsterdam accident (Crow, 1980) and the 79 workers in England (May, 1973; May, 1977) who developed chloracne had any other adverse effects; these effects were limited to asthenia. These trends have led to the conclusion that chloracne is the hallmark of TCDD poisoning (Crow, 1980). After most accidents, only patients that developed chloracne were examined further for other symptoms.

### 5.1.2.2 Porphyria Cutanea Tarda

This condition results from a disturbance in the capacity to break down hemoglobin, which leads to high levels of porphyrins in the liver and urine. Like chloracne, it is a relatively unusual condition which is not frequently encountered in other occupational situations; some cases have been shown to be inherited, rather than acquired.

Porphyria cutanea tarda was observed after the Nitro incident, and symptoms of this condition, including hirsutism and hyperpigmentation, were observed along with urine and liver changes (Suskind, 1978). After the Seveso accident, several cases of increased porphyria excretion and hyperpigmentation were observed; chromatographic examination of urine samples for levels of porphyrins excreted revealed patterns resembling mild chronic hepatic porphyria (Strik, 1979). These effects were observed only in a small portion of the exposed people in Seveso and were transient.

Porphyria cutanea tarda was not mentioned in the accounts from the other accidents. High doses of TCDD or repeated exposures may be required before this condition is observed. The possibility that concomitant exposures may have produced this condition in the Nitro and Seveso patients cannot be ruled out.

### 5.1.2.3 Hepatotoxicity

Aside from the liver function tests mentioned above, which showed transient effects immediately after exposure in workers exposed in England (May, 1973) and Seveso (Reggiani, 1978), hepatotoxicity was reported after other accidents. Hepatomegaly was observed in Nitro workers (Suskind, 1978) and Seveso residents (Bonaccorsi et al., 1978); hepatitis occurred in four of the 42 affected workers in the BASF factory (Goldmann, 1973) and hepatic disorders were mentioned (but not described) as a consequence of the accident in France in 1956 (Dugois, 1958).

Unlike chloracne, hepatotoxicity has not been reported consistently after TCDD accidents. When it is reported, it is absent in a large proportion of the people that develop chloracne. Furthermore, the different manifestations of hepatotoxicity, including hepatitis, hepatomegaly, and transient abnormal liver function tests may have resulted from different causes or as secondary or compensatory effects of TCDD exposure. It is not clear whether parameters of hepatic structure or function were even examined in the incidents that do not mention them.

TCDD treatment (see section 7.3.2.3). Rats fed diets with 2,200 ppt TCDD for 2 years showed increased serum enzyme levels as well as increased excretion of porphyrins and hepatic lesions (Kociba et al., 1978; 1979).

### 7.3.2.3 Biliary Excretion

TCDD produces adverse structural and functional effects on the biliary system. Dilation of the bile duct was observed at death in monkeys fed 500 ppt TCDD in feed for nine months (Allen et al., 1977). Proliferation and stratification of bile duct epithelium were observed in monkeys that died after subacute doses of toxic fat were administered in their feed (Allen and Carstens, 1967).

Prolonged bromsulphthalein clearance times were observed in horses after high and often lethal exposure to TCDD in salvage oil sprayed on arenas. Severe biliary cirrhosis was observed in all horses at autopsy (Case and Coffmann, 1973). Proliferation of bile ducts was observed in mice administered weekly doses of 25 ug TCDD/kg TCDD (Vos et al., 1974).

In rats that were administered 1.0 ug/kg/day of TCDD for 13 weeks, biliary hyperplasia was observed. Elevated serum alkaline phosphatase and direct bilirubin levels were also detected in these rats, indicating impairment of biliary functions. At a dose of 0.1 ug/kg/day for 13 weeks these changes were observed only in females, and at lower doses (0.01 and 0.001 ug/kg/day) these changes did not occur (Kociba et al., 1976). Chronic administration of feed with 0.001-5.0 ppb TCDD for 65 weeks produced mortality rates that were proportional to dose. Hyperplasia of the bile ducts was observed in all animals that died (Van Miller and Allen, 1977).

### 7.3.2.4 Porphyria

Porphyria is a disorder of hemoglobin metabolism. Porphyria cutanea tarda is a severe form of porphyria and has occurred in workers that were exposed to TCDD (see chapter 5). The suggested biochemical lesion in hepatic porphyria is an inhibition of the enzyme uroporphyrinogen decarboxylase. This enzyme converts uroporphyrinogen to coproporphyrinogen, intermediates in the hepatic conversion of delta-aminolevulinic acid to heme. Urinary porphyrin excretion is elevated in cases of hepatic porphyria.

In man, a relative increase in 7- and 8-carboxy-containing porphyrin metabolites over 4- to 6-carboxy-porphyrin metabolites is a more sensitive indicator of porphyria than the level of total porphyria excretion. In cases of porphyria cutanea tarda, total urinary porphyrins can be comprised of 45-70 percent uroporphyrin and 25-35 percent heptacarboxylic porphyrin, compared to much lower proportions in normal urine. Hepatic accumulation of these porphyrins occurs before clinical symptoms are manifest or urinary levels are elevated (VA, 1980).

Porphyria has been observed in male and female rats after 1 ug/kg/day of TCDD was administered for 13 weeks. Only females developed porphyria from 0.1 ug/kg/day doses for 13 weeks. Evidence of porphyria included elevated excretion of total porphyrins, uroporphyrin, and delta-aminolevulinic acid. Mortality for the higher and lower doses were 33 percent and none, respectively (Kociba et al., 1976). Porphyria was also observed in the rat after chronic dosing (Greig et al., 1979; Kociba et al., 1978; 1979).

In another study, porphyria was not observed in any rats that died after 16-31 daily doses of 10 ug/kg of TCDD or guinea pigs that died from four to five weekly doses of 1 ug/kg TCDD. In this study, porphyria was evaluated by examining the liver under ultraviolet light for red fluorescence, an indication of excess amounts of porphyrins, and this method may have been too insensitive to detect the expected increase in porphyrins (Gupta et al., 1973). No changes in delta-aminolevulinic acid synthetase levels were detected in rat livers pretreated with up to 25 ug/kg TCDD 1 day prior to sacrifice, and induction of delta-aminolevulinic acid synthetase by allylisopropylacetamide (ALA) was not affected by TCDD over 28 days. Twenty-four-hour pretreatment of fetal or neonatal rats with TCDD also failed to alter ALA synthetase activity (Woods, 1973). In these experiments, TCDD was not administered chronically or at a high dose with a latency period greater than 1 day, which is probably necessary to allow enzyme changes to become manifest.

In mice, porphyria has been observed after four weekly doses of 25 ug/kg TCDD were administered. This dosage produced a 2,000-fold increase in the hepatic levels of 7- and 8-carboxyporphyrins, which were analyzed by thin layer chromatography. Total iron content in the liver was elevated significantly (to 150 percent of the control level), delta-aminolevulinic acid synthetase activity was increased, and histopathological changes in the liver were noted. A single lethal dose of 150 ug/kg of TCDD produced a 4,000-fold increase in hepatic uroporphyrins 21-25 days after treatment. Hepatic iron levels were elevated to about 250 percent of the level of the control group (Goldstein et al., 1973). In another study, the porphyrinogenic effects of intraperitoneal doses to mice of 25 ug/kg/week for 11 weeks included a 10-fold elevation in urine porphyrin levels and a reduction to 20 percent of uroporphyrinogen carboxylase activity, compared to control mice. As described above (section 7.3.2.1), iron-deficient mice with hemoglobin levels of 5.5 g/dl showed no signs of porphyria after the same dosage of TCDD was administered (Sweeney et al., 1979; Jones and Sweeney, 1979).

## 7.4 NUTRIENT ABSORPTION AND UTILIZATION

Changes in body weight and food consumption after herbicide and TCDD treatment are described here. Factors that can affect nutrient utilization are also considered in this section. These factors include absorption of nutrients from the gut, hormonal alterations that effect regulation of nutrient utilization, and biochemical factors that reflect levels of circulating protein and lipid precursors and metabolites.

### 7.4.1 2,4-D and 2,4,5-T

Decreased weight gain has been observed in most species studied, after low subacute doses of phenoxy acids were administered. Higher doses produced complete cessation of eating in some animals (see tables 7-1 and 7-2).

VETERANS OF FOREIGN WARS OF THE UNITED STATES

THE COMMANDER-IN-CHIEF

STATEMENT OF

JAMES R. CURRIEO
COMMANDER-IN-CHIEF
VETERANS OF FOREIGN WARS OF THE UNITED STATES

BEFORE THE

SUBCOMMITTEE ON COMPENSATION, PENSION AND INSURANCE
COMMITTEE ON VETERANS' AFFAIRS
UNITED STATES HOUSE OF REPRESENTATIVES

WITH RESPECT TO

H.R. 1961, THE VIETNAM VETERANS AGENT ORANGE RELIEF ACT

WASHINGTON, D. C. APRIL 26, 1983

MR. CHAIRMAN AND MEMBERS OF THE SUBCOMMITTEE:

On behalf of the 1.96 million members of the Veterans of Foreign Wars of the United States, I welcome the opportunity to express our views to this distinguished Subcommittee regarding H.R. 1961, a bill introduced by the Honorable Thomas A. Daschle for the purpose of providing a presumption of service connection for the occurrence of certain diseases related to exposure to herbicides or other environmental hazards or conditions in veterans who served in Southeast Asia during the Vietnam era. I am pleased to note that more than 130 members of the House have chosen to co-sponsor the measure. All this support is greatly appreciated.

Three resolutions dealing with the herbicide issue were adopted by the voting delegates to our most recent National Convention in Los Angeles, California. Resolution No. 607, entitled "Herbicide Exposure," mandates that the VFW seek a rapid and accurate scientific investigation of all the aspects of this issue and that the criteria for disposition of claims be liberalized. The purpose of the resolution is to discover how all the problems related to herbicide exposure may be resolved medically and that claims for compensation for them be reasonably resolved. Resolution No. 623,

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entitled "Herbicide Fee-Basis Counseling," calls for genetic and psychological counseling to be provided to veterans who served in Vietnam and their spouses. Resolution No. 761, entitled "Agent Orange," resolves that legislation providing for compensation and health care be supported and that outreach campaigns concerning these programs be funded and encouraged at all levels of government. A copy of each of these is appended to my statement for your review.

Primarily in the last five years, Mr. Chairman, considerable national attention has been focused on Agent Orange, a defoliant used extensively in the Vietnam conflict. Since 1978, the Veterans Administration has received complaints from veterans concerning various forms of cancer, liver ailments, skin lesions, birth defects, neurological disorders, and other disorders that they attribute to exposure to Agent Orange. We, as well as others, have suggested that other causes of health problems in Vietnam be studied -- such as Agent Blue, an arsenical; Dapsone, an anti-malarial drug; Aflatoxin, a toxin occurring in Southeast Asia produced rice; and drug and alcohol abuse.

During this same time frame, Mr. Chairman, the division in the scientific community over this issue has continued; the administrative arm of the government had to be forced by law into undertaking scientific studies; the General Accounting Office (GAO) found mismanagement in the effort to get studies on the issue underway; legislation was required before an adequate level of health care was provided; arsenic compounds have been examined and known to medicine for approximately a century, and yet this information has not found its way into a review like that provided for dioxins; and finally, the questions raised by the government's offer to buy the community of Times Beach, Missouri are real to Vietnam veterans.

Mr. Chairman, I am not here to argue the scientific merits or demerits of the case for compensating Vietnam veterans due to herbicide exposure-related health problems. There are more than enough scientists available for such an exercise.

And while I do not presume to speak for them, I note that many veterans organizations, in some form, have supported the premises or enactment of this legislation.

I view it as a compassionate and responsible response on the part of the government to Vietnam veterans.

Mr. Chairman, typical of the questions and concerns that come to our headquarters in great numbers and with which I am greeted in my extensive travels throughout the country are those sentiments that came to us in a letter early in April of this year from a Jacksonville, Florida veteran:

> "AGENT ORANGE
>
> That title has been used to sell all types of newspapers and magazines from the supermarket to scientific journals.
>
> By now, most of us know that it contained Dioxin, deadly in Missouri but absolutely no threat to Vietnam veterans!
>
> Most of us also know that flight records exist that show when and where herbicides were sprayed and/or dumped; and where they were stored, shipped, moved, etc.
>
> How can we find out where and when we might have been exposed? Or put to rest our fears, knowing that most likely we were not exposed?
>
> Where is the published report or chart detailing herbicide locations and military unit locations in the same time period?
>
> We know it was sprayed in the delta and on trails and on base camp perimeters. But when?
>
> Someone knows! How can we find out?"

Mr. Chairman, I view this Subcommittee as the jury. Our government has asked its Vietnam veterans to continue to bear the legacies of their honorable service. Our government has advised its Vietnam veterans that more evidence is needed to resolve doubt. How much more evidence is needed before some of this doubt may be resolved in favor of veterans?

Our government has asked its Vietnam veterans to wait while studies can be done. How much longer is it necessary to wait?

The legislation before the Subcommittee should be enacted. It represents the beginning of another aspect of the healing process for Vietnam veterans.

How long will the jury be out?

Resolution No. 607

## HERBICIDE EXPOSURE

WHEREAS, defoliants, the most commonly known being "Agent Orange" were utilized in Vietnam; and

WHEREAS, may of this nation's Vietnam Veterans were exposed, in varying degrees to these toxic defoliants; and

WHEREAS, some researchers contend that exposure to herbicides containing dioxin cause health defects, nervous systems disorders, liver dysfunctions, genetic changes, spontaneous abortions or miscarriages, nausea, dizziness, and skin disease; and

WHEREAS, some experts contend that dioxin concerns are considerably overblown and that no medical evidence exists to substantiate compensatory claims; and

WHEREAS, these factors, as well as several industrial accidents involving dioxin, have brought about one of the nations's most heated and potentially wide-ranging controversies; now, therefore

BE IT RESOLVED, by the 83rd National Convention of the Veterans of Foreign Wars of the United States, that we use every means at our disposal to insure an accurate and timely completion of studies to resolve this question independently of the Veterans Administration; and

BE IT FURTHER RESOLVED, that we endorse and support liberalizing criteria for proper disposition of herbicide related claims.

Adopted by the 83rd National Convention of the Veterans of Foreign Wars of the United States held in Los Angeles, California, August 13-19, 1982.

Resolution No. 607

## Resolution No. 623

## HERBICIDE FEE BASIS GENETIC COUNSELING

WHEREAS, many veterans who believe they were exposed to herbicides during military service have psychological and physical disabilities affecting procreative powers; and

WHEREAS, many of these veterans have sired birth defective offspring, stillborn children and, in some cases, have wives who miscarried or fear the above may occur; and

WHEREAS, the Veterans Administration has no facilities of any consequence to assist veterans in these areas; now, therefore

BE IT RESOLVED, by the 83rd National Convention of the Veterans of Foreign Wars of the United States, that we support the institution of a Veterans Administration program of fee basis genetic and psychological counseling in all herbicides related claims in which the veteran has psychological or physical impairments of procreative power.

Adopted by the 83rd National Convention of the Veterans of Foreign Wars of the United States held in Los Angeles, California, August 13-19, 1982.

Resolution No. 623

## Resolution No. 761

## AGENT ORANGE

WHEREAS, since the dream of prevention at the beginning of the chemical age can no longer be realized, the already occurred catastrophe must be dealth with in an equitable fashion; and

WHEREAS, the herbicide code named "Agent Orange" was contaminated by the most toxi man-made substance in the world and the United States Military Personnel in Southeast Asia were grossly contaminated and exposed (in doses 300 times that of the then industry standard) and those military personnel and their offsprings are revealing the effects of that exposure; now, therefore

BE IT RESOLVED, by the 83rd National Convention of the Veterans of Foreign Wars of the United States, hereby demand that the Congress of the United States and the President of the United States, act immediately to enact and implement the provisions of House of Representatives Bill 2493, herein known as the Vietnam Veterans Agent Orange Act; and

BE IT FURTHER RESOLVED, that the United States Government subsidize and encourage the states to conduct vigorous identification, treatment and information campaigns for veterans; and

BE IT FURTHER RESOLVED, that the States be encouraged to, in turn, encourage subunits of the States, Public and Private Organizations to assist in the identification, informational dissemination and treatment of "Agent Orange" exposure.

Adopted by the 83rd National Convention of the Veterans of Foreign Wars of the United States held in Los Angeles, California, August 13-19, 1982

Resolution No. 761

VETERANS OF FOREIGN WARS OF THE UNITED STATES

THE COMMANDER-IN-CHIEF

VR

May 2, 1983

The Honorable Douglas Applegate
Chairman
Subcommittee on Compensation,
  Pension and Insurance
Committee on Veterans' Affairs
U. S. House of Representatives
Washington, DC 20515

Dear Mr. Applegate:

Thank you for the opportunity to respond to follow-up questions for the record regarding H.R. 1961, The Vietnam Veterans Agent Orange Relief Act.

Our responses to the questions posed are as follows:

Question #1 - How does your position in support of this legislation square with your Resolution 607 which mandates scientific investigation of all aspects?

Response #1 - Resolution 607 does not presume resolution of all scientific questions concerning exposure to herbicides by American troops during their service in Southeast Asia will occur simultaneously. The language found in H.R. 1961 is not intended to preclude further scientific investigation of all aspects of this issue. In our view, the language in the bill suggests further studies.

Question #2 - Do you think presumptive service connection should be limited to a select group of veterans?

Response #2 - Presumptive service-connection, in our view, should be granted to those whom the Congress determines the circumstances warrant. The process of determination necessarily involves receiving information and views from agencies, scientists and the public.

Question #3 - Why should all veterans, except those who served in Vietnam, get only a year presumption for soft tissue sarcoma while Vietnam veterans get a lifetime?

Response #3 - We do not believe all presumption inequities have been eliminated from the law. In this case, we believe the Congress has the opportunity

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to accomplish the elimination of one. Clearly, the language in H.R. 1961, in our view, does not grant a lifetime presumption. However, the language in the bill does not preclude the Congress, if it desires, from doing so.

Question #4 - Do you think presumptions should be open-ended or should there be a reasonable relationship between the period of service and the time when the disability is first noted?

Response #4 - We agree a reasonable relationship should exist, but the relationship should be one that is reasonably and scientifically arrived at. It is the responsibility of the Congress, through the application of its investigative and legislative powers, to make such determinations.

Question #5 - What scientific evidence do you have to support H.R. 1961? How did you arrive at the conclusion that this bill meets your convention mandate?

Response #5 - Materials relevant to this issue were supplied to the Subcommittee for review prior to the hearing. While the VFW does not maintain research capability, we do review research work accomplished by others, including the VA. Two of the three conditions in the bill were acknowledged by the VA on April 26, 1983 in the VA Administrator's testimony. We are not aware of evidence that would refute the third.

With best wishes and kind regards, I am

Sincerely,

James R. Currieo

JAMES R. CURRIEO
National Commander-in-Chief

*Veterans of the Vietnam War*

STATEMENT OF
DONALD J. SWARTZ
NATIONAL DIRECTOR OF VETERANS AFFAIRS
BEFORE THE
SUBCOMMITTEE ON COMPENSATION, PENSION AND INSURANCE
UNITED STATES HOUSE OF REPRESENTATIVES
APRIL 26, 1983

MR. CHAIRMAN AND MEMBERS OF THE SUBCOMMITTEE:

I AM HONORED TO APPEAR BEFORE THE SUBCOMMITTEE ON COMPENSATION, PENSION AND INSURANCE TO DISCUSS MY VIEWS ON HOUSE BILL 1961, WHICH YOU HAVE INTRODUCED MR. CHAIRMAN.

THIS BILL WOULD AMEND TITLE 38 OF THE UNITED STATES CODE, TO PROVIDE A PRESUMPTION OF SERVICE CONNECTION FOR THE OCCURENCE OF CERTAIN DISEASES RELATED TO EXPOSURE TO HERBICIDES OR OTHER ENVIRONMENTAL HAZARDS OR CONDITIONS IN VETERANS WHO SERVED IN SOUTHEAST ASIA DURING THE VIETNAM ERA.

I AM A FOUR YEAR VETERAN OF THE UNITED STATES NAVY OF WHICH THE LAST TWO YEARS WERE SPENT AS A CORPSMAN ATTACHED TO THE MARINE CORP. EIGHT MONTHS OUT OF THE LAST TWO YEARS WAS SERVED IN VIETNAM.

FIRST OF ALL, I WOULD LIKE TO TAKE THIS OPPORTUNITY TO SINCERELY THANK MR. DASCHLE FOR INTRODUCING HOUSE BILL 1961 AND THE ONE HUNDRED THIRTEEN CO-SPONSORS FOR HAVING THE COURAGE AND CONCERN FOR THE VIETNAM VETERAN, TO TACKLE SUCH AND EMOTIONAL AND CONTROVERSIAL ISSUE.

THE CENTER FOR DISEASE CONTROL HAS RATED ANY LEVEL OF DIOXIN ABOVE ONE PART PER BILLION AS POSING A SERIOUS RISK TO HEALTH. THOUSANDS OF VIETNAM VETERANS FOR YEARS HAVE FLOCKED TO THE VETERANS ADMINISTRATION COMPLAINING OF A MYRIAD OF PROBLEMS, YET THESE COMPLAINTS HAVE FALLEN ON DEAF EARS. THE VIETNAM VETERAN HAS BEEN SLAPPED IN THE FACE ENOUGH - HOW MANY MORE TIMES MUST WE TURN THE OTHER CHEEK? WE HAVE SEEN THE ENVIRONMENTAL PROTECTION AGENCY ISSUE A BAN ON THE PRODUCTION OF 2-4-5-T WHICH PRODUCED THE DIOXIN IN AGENT ORANGE. WE HAVE SEEN THE ENTIRE COMMUNITY OF TIMES BEACH, MISSOURI EVACUATED AT GOVERNMENT EXPENSE BECAUSE OF EXPOSURE TO DIOXIN, YET THE VETERANS ADMINISTRATION PERSISTS IN ITS HARD LINE; THAT THERE IS NO PROOF THAT DIOXIN IS HARMFUL TO ONE'S HEALTH. HOW CAN IT BE THAT DIOXIN IS HARMFUL TO THE PEOPLE IN TIMES BEACH, WHERE THE LEVELS OF DIOXIN WERE FAR LESS THAT WHAT WE WERE EXPOSED TO, AND YET NOT HARMFUL TO THE VIETNAM VETERAN? WE WERE NOT INNOCULATED AGAINST THIS TOXIN, NOR WERE WE INFORMED OF THE POSSIBLE ADVERSE HEALTH EFFECTS, AND WE WERE NOT ISSUED PROTECTIVE CLOTHING.

ON AUGUST 27, 1982, WE AGAIN HAD TO TURN THE OTHER CHEEK. THE NEW YORK TIMES PUBLISHED AN ARTICLE CONCERNING FOURTY-SEVEN RAILROAD WORKERS WHO WERE EXPOSED TO DIOXIN. THEY WERE AWARDED $58 MILLION DOLLARS IN SETTLEMENT. THESE WORKERS DEVELOPED MEDICAL PROBLEMS INDENTICAL TO THOSE THE VIETNAM VETERANS HAVE BEEN COMPLAINING ABOUT. THE AMOUNT OF DIOXIN THESE WORKERS WERE EXPOSED TO WAS 22 PARTS PER BILLION, AGAIN, FAR, FAR LESS THEN THE LEVEL OF DIOXIN IN AGENT ORANGE. BARELY A DAY GOES BY THAT ONE DOESN'T READ SOMETHING ABOUT A COMMUNITY OR WORKERS BEING EXPOSED TO DIOXIN, AND THE CONCERN THAT THEIR EXPOSURE INVOKES.

YET WHERE IS THE CONCERN FOR THE VIETNAM VETERAN? JUST AS THE VIETNAM VETERANS MEMORIAL DEDICATED LAST NOVEMBER WAS A STEP IN THE RIGHT DIRECTION TO FINALLY RECOGNIZE THE SACRIFICE OF THE VIETNAM VETERAN, SO TO IS HOUSE BILL 1961 A STEP IN THE RIGHT DIRECTION: BUT ONLY A STEP IN WHAT WILL BE A LONG, LONG JOURNEY.

ON PAGE THREE OF THIS BILL, IT ITEMIZES THE CONDITIONS THAT WOULD BE GRANTED PRESUMTION FOR SERVICE CONNECTION, AND I DEFINITELY CONCUR WITH THIS JUDGEMENT. HOWEVER, WHAT DO WE TELL THOSE VIETNAM VETERANS WHO ARE SUFFERING FROM OTHER MEDICAL CONDITIONS KNOWN TO BE INDUCED BY EXPOSURE TO DIOXIN? DO WE ONCE AGAIN ASK THEM TO TURN THE OTHER CHEEK? I IMPLORE YOU TO EXPAND THIS BILL TO INCLUDE THESE OTHER KNOWN MEDICAL CONDITIONS.

ITEM (2) ([illegible]) (IV) OF THIS BILL WOULD ALLOW THE ADMINISTRATOR OF THE VETERANS ADMINISTRATION TO INCLUDE OTHER DISEASES THAT MEDICAL RESEARCH SHOWS CAUSES HEALTH PROBLEMS. I HAVE SERIOUS RESERVATIONS WITH THIS PROTION OF HOUSE BILL 1961. IN VIEW OF THE HARD LINE APPROACH OF THE VETERANS ADMINISTRATION HANDLING OF THE AGENT ORANGE ISSUE, I MUST SAY THAT I VIEW THEIR HAVING THE FINAL SAY ON WHAT DISEASES WILL OR WILL NOT BE INCLUDED, WITH MUCH SKEPTICISM. IF ONE FEELS THIS SKEPTICISM IS UNWARRANTED, THEN I SUGGEST YOU READ THE GENERAL ACCOUNTING OFFICE REPORT TO CONGRESS DATED OCTOBER 25, 1982 REGARDING THE VETERANS ADMINISTRATION'S HANDLING OF THE AGENT ORANGE ISSUE. THEREFORE, I SUGGEST THAT THE CENTER FOR DISEASE CONTROL OR SOME OTHER AGENCY EVALUATE THE KNOWN LITERATURE ON DIOXIN AND IT'S ADVERSE HEALTH EFFECES, AND DETERMINE WHAT OTHER DISEASES SHOULD BE GRANTED SERVICE CONNECTION, AND THAT THE VETERANS

ADMINISTRATION BE MANDATED TO ACCEPT THEIR FINDINGS AND GRANT DISABILITIES TO THOSE THAT QUALIFY.

ONE HUNDRED AND SIX THOUSAND VETERANS HAVE TAKEN THE AGENT ORANGE EXAM AT VETERANS ADMINISTRATION HOSPITALS THROUGHOUT THE COUNTRY. THAT CERTAINLY IS A LARGE ENOUGH POPULATION TO DRAW CONCLUSIONS ABOUT VARIOUS HEALTH PROBLEMS. IF IT IS OBVIOUS THAT CERTAIN TRENDS DEVELOPED, THEN WHY MUST WE WAIT UNTIL 1991 FOR ACTION. AGAIN, IF THE ONLY COMMON FACTOR IS SERVICE IN VIETNAM, THEN THE ONLY CONCLUSION THAT CAN BE REACHED IS THAT IT MUST BE A SERVICE CONNECTED DISABILITY.

IN MY POSITION AS DIRECTOR FOR VETERANS AFFAIRS FOR THE VETERANS OF THE VIETNAM WAR AND AS DIRECTOR OF ONE OF PENNSYLVANIA'S GOVERNOR'S VETERANS OUTREACH AND ASSISTANCE CENTERS, I HAVE LITERALLY SEEN THOUSANDS OF VIETNAM VETERANS. I HAVE DEALT WITH THEM ON A DAILY BASIS FOR SEVERAL YEARS AND I CAN ASSURE YOU THAT THEIR COMPLAINTS AND CONCERNS ARE NOT UNFOUNDED. I HAVE SEEN YOUNG MEN DIE OF CANCER AND OTHER DISEASES. I HAVE SEEN THE BIRTH DEFECTS IN OUR CHILDREN. I HAVE SEEN THOSE MENTALLY TORTURED BECAUSE OF WHAT THEY DID OR SAW WHILE IN SERVICE TO THEIR COUNTRY. I HAVE MET VETERANS FROM ALL OVER THE COUNTRY WHO ARE HAVING PROBLEMS AND THERE IS ONLY ONE THING THEY HAVE IN COMMON. THAT GENTLEMEN, IS THAT THEY SERVED THEIR COUNTRY IN VIETNAM.

ON BEHALF OF MY BROTHERS, I WANT TO THANK THIS COMMITTEE FOR GIVING ME THE OPPORTUNITY TO PRESENT MY VIEWS. THANK YOU.

*Veterans of the Vietnam War*

STATEMENT OF
MICHAEL MILNE
NATIONAL DIRECTOR
VETERANS OF THE VIETNAM WAR
BEFORE THE
SUBCOMMITTEE ON COMPENSATION, PENSION AND INSURANCE
UNITED STATES HOUSE OF REPRESENTATIVES
APRIL 26, 1983

MR. CHAIRMAN AND MEMBERS OF THE SUBCOMMITTEE:

THANK YOU FOR THE OPPORTUNITY TO PRESENT THE VIEWS OF THE VETERANS OF THE VIETNAM WAR WITH RESPECT TO HOUSE BILL 1961. MY NAME IS MICHAEL MILNE. I AM THE NATIONAL DIRECTOR OF THE VETERANS OF THE VIETNAM WAR, A FRATERNAL ORGANIZATION THROUGHOUT THE UNITED STATES, AND WE ARE STILL GROWING. I AM A VIETNAM VETERAN WHO SERVED IN THE UNITED STATES ARMY FOR SEVEN YEARS. I AM A VICTIM OF HERBICIDE EXPOSURE. I MUST WEAR BRACES ON BOTH HANDS, A BACK BRACE, A NECK BRACE, A PORTABLE TENS UNIT ON MY ARMS, PHOTO-GRAY LENSES AND I WALK WITH THE AID OF A CANE. I HAVE BEEN IN PHYSICAL THERAPY SINCE 1980. THE VETERANS ADMINISTRATION HOSPITAL AND PRIVATE PHYSICIANS DON'T KNOW WHAT IS WRONG WITH ME, EXCEPT FOR THE FACT I HAVE RASHES THEY CANNOT DIAGNOSE, DIARRHEA SINCE 1968, CHRONIC EPICONDYLILUS, SEVERE CHRONIC FIBROSITIS, DISCOGENIC DEGENERATIVE CERVICAL NECK DISEASE, RESPIRATORY PROBLEMS, URINARY TRACT DISORDER, POLYMYOSTIS, POLYTENDENITIS, UNSPECIFIED NEUROLOGICAL CONDITIONS, HEARING AND EYE CONDITIONS AND SINUSITIS. I WAS IN PERFECT HEALTH WHEN I WENT TO VIETNAM. MY PHYSICAL PROBLMES STARTED IN VIETNAM AND CONTINUE TO THIS DAY.

"People Who Care"

MANY STUDIES HAVE BEEN INITIATED, MANY STUDIES HAVE BEEN THROUGHLY DOCUMENTED, BUT NONE HAVE RECEIVED THE TOTAL ACCEPTANCE NECESSARY TO ENABLE YOUR VIETNAM VETERANS TO BE JUSTLY, UNQUESTIONABLY AND DESERVEDLY RECOGNIZED AS DISABLED. TELL US, HOW MUCH LONGER WILL WE HAVE TO CONTINUE TO BEAR THE BURDEN OF PROOF. YOU ARE HERE TODAY TO DECIDE THAT. THE FACT THAT IT HAS NOT BEEN DECIDED BY OUR GOVERNMENT HOW TOXIC DIOXIN IS, PREVENTS VIETNAM VETERANS FROM GETTING PROPER TREATMENT AND COMPENSATION. NO ONE DISPUTES THE FACT THAT HERBICIDES WERE USED. WHAT IS DISPUTED, IS THE LEVEL OF THE HERBICIDE'S TOXICITY. WHAT THE VETERANS ADMINISTRATION DEMANDS IS CONCLUSIVE EVIDENCE. WELL, CONCLUSIVE EVIDENCE MEANS WITHOUT THE SLIGHTEST DOUBT, AND THAT COULD BE CLOSE TO IMPOSSIBLE TO PROVIDE, SINCE IT TAKES ONE HELL OF A LOT OF THE ABSOLUTE BEST EVIDENCE TO FORCE SOMEONE (SOMEONE WHO IS DEDICATED TO ARGUING THE POINT IN THE FIRST PLACE, FOR HIS OWN WELFARE) TO TOTALLY ACCEPT IT AS FACT. IT IS AS THOUGH THEY ARE ATHEISTS OR AGNOSTICS REQUIRING US TO PROVE BEYOND A SHADOW OF A DOUBT THAT GOD DOES EXIST. AT LEAST IT TAKES ON THAT DEGREE OF DIFFICULTY TO US.

DIOXIN IS THE MOST TOXIC ORGANIC COMPOUND KNOWN, PRODUCING A WIDE RANGE OF ORGAN AND METABOLIC DYSFUNCTIONS. THERE IS A GENERAL CONSISTENCY BETWEEN THE PATTERN OF CHRONIC TOXICITY INDUCED IN ANIMALS BY DIOXIN AND DIOXIN-CONTAMINATED CHLOROPHENOLIC COMPOUNDS AND THOSE OBSERVED IN EXPOSED HUMAN POPULATIONS. SUCH TOXICITY INCLUDES: CHRONIC HEPATIC DYSFUNCTIONS, DISTURBANCES IN LIPID AND

*Veterans of the Vietnam War*

PORPHYRIN METABOLISM, NEUROLOGICAL ABNORMALITIES, NEUROLOGICAL DISORDERS, GLANDULAR DISORDERS AND DISEASES, AND BIRTH DEFECTS, MISCARRIAGES AND STILL BIRTHS. HOW MUCH OF A WARNING DO YOU NEED? THE WOMEN IN OREGON WERE STUDIED FOR FIVE YEARS. BIRTH DEFECTS COINCIDED WITH THE USE OF DIOXIN WITHOUT A DOUBT. LOOK AT VIETNAM VETERANS: DON'T YOU THINK IT A LITTLE SUSPICIOUS THAT VETERANS WHOSE AVERAGE AGE IS ONLY THIRTY-FIVE ARE SUFFERING FROM CANCERS, OTHER SEVERE METABOLIC AND PHYSICAL DYSFUNCTIONS AND PRODUCING DEFORMED CHILDRED AT RATES FAR AND ABOVE "PREDICTED AVERAGES;" WHO ARE EXPERIENCING ILLNESSES MORE PROPERLY ASSIGNED TO MUCH OLDER PEOPLE? DON'T YOU FIND THAT CURIOUS? AND WHAT IN GOD'S NAME DO VIETNAM VETERANS, PEOPLE ON FARMS IN OREGON, THE STREETS IN TIMES BEACH, MISSOURI, FACTORY WORKERS IN ITALY, CLEAN-UP WORKERS IN STURGEON, MISSISSIPPI HAVE IN COMMON? HOW ABOUT CONSIDERING EXPOSURE TO DIOXIN? HOW MUCH MORE DO YOU NEED BEFORE YOU START TO WONDER? I STAND HERE BEFORE YOU AS AN EXAMPLE OF A HERBICIDE VICTIM. I'VE TRIED TO EXPRESS SOME VITAL STATISTICS AND TO SHOW YOU THE HUMAN SIDE.

I WOULD LIKE TO THANK MR. DASCHLE AND THE OTHER CO-SPONSORS FOR TAKING THE TIME AND EFFORT AND SHOWING THEIR CONCERN FOR THE PLIGHT OF THE VIETNAM VETERAN. I AM ASKING THAT YOU EXPAND HOUSE BILL 1961 TO INCLUDE THE DISEASES THAT HAVE BEEN AFOREMENTIONED. MY BROTHERS AND I THANK YOU FOR ALLOWING ME TO TESTIFY.

"People Who Care"

*Veterans of the Vietnam War*

STATEMENT OF
JAMES ROXBY
PA STATE DIRECTOR
VETERANS OF THE VIETNAM WAR
BEFORE THE
SUBCOMMITTEE ON COMPENSATION, PENSION AND INSURANCE
UNITED STATES HOUSE OF REPRESENTATIVES

APRIL 26, 1983

MR. CHAIRMAN AND MEMBERS OF THE SUBCOMMITTE:

MY NAME IS JAMES ROXBY. I AM ONE OF THE ORIGINAL FOUNDERS OF THE VETERANS OF THE VIETNAM WAR AND AM INVOLVED IN THE NATIONALIZATION OF THE ORGANIZATION. I AM A VIETNAM VETERAN. I SPRAYED AGENT ORANGE IN VIETNAM. I CARRIED IT ON MY BACK AND SPRAYED IT. I WAS HOSPITALIZED IN VIETNAM AFTER BEING INADVERTENTLY SPRAYED. IT'S IN MY RECORDS RIGHT HERE.

HOUSE BILL 1961 WILL HELP A FEW VIETNAM VETERANS THE WAY IT IS NOW STRUCTURED, BUT IT WON'T HELP ME AND OTHER NAM VETS IN MY CONDITION. I'M DYING!

ANY QUESTIONS?

"People Who Care"

## STATEMENT OF
## STEPHEN L. EDMISTON
## ASSOCIATE DEPUTY
## NATIONAL LEGISLATIVE DIRECTOR
## DISABLED AMERICAN VETERANS
## BEFORE THE
## SUBCOMMITTEE ON COMPENSATION, PENSION & INSURANCE
## OF THE
## HOUSE COMMITTEE ON VETERANS AFFAIRS
## APRIL 26, 1983

MR. CHAIRMAN AND MEMBERS OF THE SUBCOMMITTEE:

On behalf of the more than 760,000 members of the Disabled American Veterans, I wish to thank you for the invitation to participate in today's legislative hearing on the proposed "Vietnam Veterans Agent Orange Relief Act"--H.R. 1961.

As a veterans' organization Congressionally chartered for the sole purpose of aiding and assisting those veterans who were injured or otherwise disabled during wartime service to their country, the DAV has, as a matter of priority, a very special interest in the important issues raised by the legislation pending before the Subcommittee today.

### H.R. 1961

Introduced on March 8, 1983 by a distinguished member of this Subcommittee, Mr. Daschle, H.R. 1961 proposes, through appropriate amendment to Title 38, U. S. Code, "to provide a presumption of service-connection for the occurrence of certain diseases related to exposure to herbicides or other environmental hazards or conditions in veterans who served in Southeast Asia during the Vietnam Era."

Section 2 of the bill sets forth Congressional concern with respect to the possible adverse health effects and other related problems, including birth defects in the offspring of veterans who served in Southeast Asia during the Vietnam Era, which may have arisen as a result of their exposure to the various phenoxy herbicides used during that time period.

By appropriate amendment to Section 312 of Title 38, U. S. Code, Section 3 of the bill proposes to add a new subsection that would, for the purposes of Section 310 and subject to the provisions of 313, 38 USC, establish a presumption of service-connection for:

1. Soft tissue sarcomas;
2. Porphyria cutanea tarda;
3. Active and residual chloracne and chloracne form lesions;
4. Any diseases the VA determines are the result of exposure to the various phenoxy herbicides;

in the cases of veterans who suffer from such diseases and who, served in Southeast Asia during the Vietnam Era.

Finally, the presumption of service-connection established by this Section of the bill would terminate one year after the Veterans Administration has submitted the first report required by Section 307(b)(2) of the Veterans Health Program's Extension and Improvement Act of 1979 (Public Law 96-151)--which requires the VA to conduct an epidemiological study of veterans serving during the Vietnam Era to determine if they had suffered long-term adverse health effects from exposure to Agent Orange, or other herbicides, chemicals, medications or environmental hazards or conditions.

The bill, as proposed, does not set forth a specific effective date, therefore, it would become effective upon date of enactment.

* * *

Mr. Chairman, the possible adverse health effects of exposure to the herbicide known as Agent Orange is and has been of great concern to Vietnam Era veterans, their families and the general public. As you are well aware, thousands of Vietnam veterans were exposed to this phenoxy herbicide by virtue of the widespread use of the defoliant by the military in Vietnam--especially during the period 1965 through 1970.

Serious questions have been raised concerning the relationship between exposure to Agent Orange and more specifically, exposure to dioxin--the toxic element in Agent Orange--and the subsequent development or appearance of such impairments as cancer, birth defects, nervousness, skin conditions, numbness of extremities and paralysis, miscarriages, etc.

As a result of this concern, Congress directed (P.L. 96-151) the VA to design a protocol for and conduct an epidemiological study of persons who were exposed to Agent Orange.

Subsequent legislation, Public Law 97-72, expanded the scope of this study to include an evaluation of the adverse health effects which may result from other factors (in addition to Agent Orange exposure) including exposure to other herbicides, chemicals, medications or environmental hazards or conditions. In addition, the legislation also permitted the inclusion of other studies and literature relating to adverse health effects caused by Vietnam service or adverse health effects caused in comparable situations, within the scope of the VA review.

Further, Public Law 97-72 extended eligibility for VA health care services to Vietnam Era veterans for the treatment of disabilities that may be related to exposure to dioxin or other toxic substances found in herbicides or defoliants used in Vietnam.

As you know, Mr. Chairman, the study mandated by Public Law 96-151 was initially embroiled in litigation and has not moved forward in a manner which we all would have preferred. Subsequently, due to the urging of many members of Congress and particularly the Chairman and Ranking Minority Member of the House Veterans Affairs Committee the Veterans Administration agreed to transfer the study to the Centers for Disease Control. VA and CDC entered into such an agreement in January of this year.

More than three years have passed since Congress first authorized the VA to conduct a study seeking answers to the serious questions raised regarding exposure to Agent Orange. Today, Vietnam Era veterans and their families are no closer to having their questions answered than they were in 1979.

Because we too share the concerns of Vietnam Era veterans and their families over the possible effects exposure to Agent Orange may have had upon their health, Mr. Chairman, the DAV is appalled, as are most Vietnam Era veterans, by the lack of decisive action on the part of the VA to complete this study.

There is no question of the DAV's sincerity and concern for the well being of those men and women who served as members of our Armed Forces in Vietnam--our track record speaks for itself.

Quite frankly though, Mr. Chairman, the DAV has some serious reservations regarding the legislative proposal (H.R. 1961) pending before the Subcommittee today.

Initially, I must point out that our membership has mandated, as the result of a Resolution (No. 024) adopted at our most recent National Convention, that the DAV support "...the enactment of legislation which would extend an open-ended presumption of service-connection for any identifiable residual disability or disease incurred by veterans which is scientifically proven to be the result of exposure to the chemical defoliant Agent Orange or any other phenoxy herbicide."

To date, no diseases or disabilities in humans, with the exception of the skin condition known as chloracne (which is apparently under question by some in the scientific community at this time), have been scientifically proven to be the result of exposure to the chemical defoliant Agent Orange or any other phenoxy herbicide.

We recognize, Mr. Chairman, that the pending legislation is a sincere effort to make up for the time that has been lost in completing the study mandated by Public Law 96-151.

However, we must seriously question the impact H.R. 1961 will have upon the integrity of the VA's Disability Compensation Program--a creative system that has served all categories of veterans who have been disabled as a result of their military service very well for more than 60 years.

In our view, the pending bill deviates from the traditional methods of establishing, on a factual basis, the incurrence or aggravation of a disease or disability during active duty military service, or as a direct result of such service. We believe such a departure from the VA's current rules of evidence could, in fact, undermine the integrity of our present Disability Compensation Program. No longer would accepted medical principles be the basis in law for establishing presumptions of service-connection for various diseases and disabilities.

Moreover, as proposed, H.R. 1961 would extend, in addition to the service-connected disability compensation benefits, the full range of benefits that accrue to all otherwise eligible service-connected disabled veterans and their dependents.

For example, depending upon the degree of disability and other eligibility criteria, the following benefits could accrue to the eligible veteran and his dependents: (1) a varying range of health care benefits, including potential CHAMPVA eligibility for dependents; (2) Chapter 31 vocational rehabilitation educational benefits; (3) dependency allowances; (4) National Service Life Insurance; (5) certain housing and automobile assistance benefits; (6) preference in federal and state employment; (7) DIC and dependents educational assistance, as well as other benefits.

Aside from these reservations and in light of the sunset provisions of the pending bill, we are also deeply concerned with the potential impact of this legislation upon the lives of those Vietnam Era veterans and their families who would become eligible for all the benefits that would accrue from the bill--only to lose all entitlement upon completion of the study, particularly so, should there be no evidence found on which to base service-connection for these claimed impairments.

We realize that the bill's sponsors anticipate legislative recommendations and Congressional action upon receipt of the completed study mandated by Public Law 96-151. However, Mr. Chairman, what action will this Subcommittee and indeed, the entire Congress take, should the completed study conclude, to the satisfaction of the scientific community, that veterans exposed to phenoxy herbicides in Southeast Asia suffer no greater health problems than any other segment of the population?

With these concerns in mind, Mr. Chairman, we believe the pending bill does contain a provision that would, as Mr. Daschle is attempting to do, compel the Veterans Administration to move forward and take positive steps to equitably consider and resolve the pending disability claims filed by Vietnam Era veterans relating to phenoxy herbicide exposure.

While this provision is a step in the right direction, we do not favor the enactment of legislation that would place the burden of establishing such presumptions upon the Veterans Administration. Historically, this is a function of Congress as generally outlined in Sections 301 and 312 of Title 38, U.S. Code.

However, it appears that the intent of this portion of Section 3 of H.R. 1961 is to mandate the development of a specific method for the disposition of claims related to herbicide exposure.

In this connection, Mr. Chairman, we would strongly recommend, in light of the findings contained in the GAO Report--Health Effects Of Exposure To Herbicide Orange In South Vietnam Should Be Resolved--dated April 6, 1979, that the VA be required to develop concise and in-depth guidelines for the disposition of phenoxy herbicide-related claims since (as reported by GAO) the toxic effects of such herbicides has been reported by humans exposed during industrial accidents involving the production of such chemicals.

In our view, such guidelines should embrace: (1) the fact that the veteran served in Southeast Asia during the time phenoxy herbicides were in use by the military services; (2) the veteran is suffering from a condition which would be compensably disabling; (3) strict adherence to the provisions of Section 313, Title 38, United States Code regarding the absence of any other intervening cause which may have led to the veteran's present disability; (4) a measure of the level of dioxin retained in the human body which, in and of itself, would be considered the probable cause of the present disease or disability; (5) incorporate all known health effects of phenoxy herbicide exposure as reported by GAO in their April 6, 1979 report, pages 17 and 18, which sets forth a variety of dermatological, internal, neurological and psychiatric disorders; and (6) the relationship of acute symptoms appearing immediately following exposure to more chronic, latent conditions.

Mr. Chairman, the development and implementation of such guidelines will go a long way toward insuring the integrity of the VA's disability compensation program, as well as establishing a formal procedure for the disposition of those disability claims attributed to phenoxy herbicide exposure in Southeast Asia.

This approach, in preparation of obtaining the results of the study mandated by Public Law 96-151, is by no means new. Similar guidelines were developed for claims filed by former prisoners of war--the VA adjudicated cases based upon specific guidelines until Congress received definitive study results. Subsequently, legislation was initiated and enacted into law which generally liberalized the various VA benefits and services provided to former prisoners of war.

Mr. Chairman, I trust our views on this most important and emotional subject are received in the same manner as we have presented them--with true concern and sincerity for those who served in our nation's Armed Forces during the Vietnam Era.

That concludes my testimony, Mr. Chairman. I again wish to thank you and the members of the Subcommittee for permitting us to provide our views on this most important subject.

* * *

RESOLUTION NO. 024
LEGISLATIVE

## TO EXTEND A PRESUMPTION OF SERVICE-CONNECTION FOR RESIDUAL DISABILITY RELATED TO AGENT ORANGE

WHEREAS, from 1965 to 1970 some 10.65 million gallons of the chemical defoliant Agent Orange was used extensively throughout Vietnam; and

WHEREAS, thousands of U.S. Troops were sprayed directly or were repeatedly exposed to herbicides containing dioxin; and

WHEREAS, with the exception of a skin condition -- chloracne -- the Veterans Administration has not granted a single claim for residual disabilities related to exposure to Agent Orange; and

WHEREAS, Public Law 96-151 mandated the Veterans Administration to conduct a study of the long term health effects on veterans exposed to the variety of phenoxy herbicides used in Vietnam; and

WHEREAS, the lack of conclusive scientific evidence has continued to hamper the claims of veterans who relate their residual disability to their exposure to the herbicide Agent Orange; NOW

THEREFORE, BE IT RESOLVED that the Disabled American Veterans in National Convention assembled in Las Vegas, Nevada, July 18-22, 1982, seeks the enactment of legislation which would extend an open-ended presumption of service-connection for any identifiable residual disability or disease incurred by veterans which is scientifically proven to be the result of exposure to the chemical defoliant Agent Orange or any other phenoxy herbicide.

* * *

PVA
PARALYZED
VETERANS
OF
AMERICA
CHARTERED BY THE CONGRESS OF THE UNITED STATES

STATEMENT OF
R. JACK POWELL, EXECUTIVE DIRECTOR
PARALYZED VETERANS OF AMERICA
BEFORE THE
SUBCOMMITTEE ON COMPENSATION, PENSION AND INSURANCE
OF
THE HOUSE COMMITTEE ON VETERANS' AFFAIRS
CONCERNING
H. R. 1961, THE "VIETNAM VETERANS AGENT ORANGE RELIEF ACT"
APRIL 26, 1983

Mr. Chairman and Members of the Subcommittee, Paralyzed Veterans of America appreciates this opportunity to express its views on the "Vietnam Veterans Agent Orange Relief Act," H.R. 1961. PVA's members are all veterans who have experienced spinal cord injury or dysfunction. Many of our members served in Southeast Asia during the Vietnam War and have expressed deep, personal concerns regarding the possible adverse health effects of exposure to Agent Orange and other herbicides used there.

PVA applauds the efforts of the distinguished sponsor and the many cosponsors of this legislative proposal. The recognition of the potential health hazards of exposure to dioxins and other chemical agents used in Southeast

801 Eighteenth St., N.W., Washington, D.C. 20006 (202) USA-1300

Asia and the problems experienced by veterans who served there are areas which must be continually addressed and reviewed by the government.

PVA, however, does not support the passage of the provisions of H.R. 1961. It is our position that the preseumption of service-connected disabilities must be related to sound, reasonable medical evidence that such a connection exists. The diseases specifically enumerated in H.R. 1961, soft-tissue sarcomas, porphyria cutanea tarda, and active and residual chloracne and chloracne form lesions do not meet this test.

In the case of chloracne, symptoms of the disease are manifest shortly after contact with chlorine compounds such as those used in Southeast Asia. In the several cases where service-connection has been provided to veterans with chloracne lesions, it is our understanding, that the condition was readily apparent while the individuals were still in service.

Mr. Chairman, it is certainly not our position to deny veterans due compensation for diseases or injuries incurred while serving the nation. PVA strongly believes it is the nation's obligation to provide for veterans who have experienced health problems related to their service. However, the three specific diseases for which the presumption of service-connection would be provided by H.R. 1961 do not meet the criteria established for reasonable medical evidence.

The language of Section 2(1) of H.R. 1961 causes PVA concern. The term "may be the result of exposure" is extremely broad as are the potential conditions and causative agents or factors listed. Such nonspecific language can only reinforce the doubts and anxieties of veterans who served in Southeast Asia.

This language is positive in that it clearly indicates the growing recognition that adverse health effects experienced by veterans should be viewed and studied in the context of the entire Vietnam experience. Hopefully, this growing recognition will be transmitted and related to the studies by the scientific community. The Center for Disease Control (CDC) has already indicated that their epidemiological study will go beyond the limited scope of exposure to the herbicide, Agent Orange, and will investigate the multifaceted environment of Southeast Asia in which veterans served.

Another area of concern to PVA is the broad authority extended by H.R. 1961 to the Administrator of Veterans Affairs. Section 3(B) would give the Administrator the ability to determine the presumption of service-connection for other diseases and conditions related to service in Southeast Asia. PVA believes that Congress should not provide such broad authority to the Administrator, but rather should maintain this authority. Historically, Congress has determined, legislatively, the presumption of service-connection as medical evidence has become available.

Mr. Chairman, PVA strongly supports efforts to finally resolve the potential adverse health effects related to service in Southeast Asia. We commend this Subcommittee for continually addressing this issue and with attempting to address the needs of veterans who served in Southeast Asia.

While PVA cannot support passage of H.R. 1961, we do view this effort as a positive action in focusing attention on this continuing problem. We believe that veterans must be provided as much information as is available and in as timely a manner as possible on any health related problems resulting from service in Southeast Asia. The commitment of the Congress and the Members of the Committees on Veterans' Affairs has been instrumental in providing such information. It is extremely important that methods be devised, however, to improve the provision of such information to veterans. The psychological burden of the uncertainties associated with unknown health problems related to military service may be as detrimental to veterans' well-being as the actual service in Southeast Asia. It is our hope that this Subcommittee and the sponsors of H.R. 1961 will work to ensure that veterans are provided all available data resulting from research on the health effects of service in Vietnam and Southeast Asia.

Additionally, PVA is well aware of the problems associated with medical research, both in terms of time and resource requirements. We ask that this Subcommittee and the Congress work to ensure that all necessary resources be made available for research and studies on the health-effects of veterans' service in Vietnam. We also request that interim reports from these ongoing efforts be made available to the public and, specifically, to veterans who served in Southeast Asia.

Mr. Chairman and Members of the Subcommittee, again on behalf of the members of Paralyzed Veterans of America, I wish to express my appreciation for this opportunity to express our views on H.R. 1961. PVA continues to support the provision of a presumption of service connection for veterans with diseases and conditions when reasonable medical evidence has determined the etiology is service related.

This concludes my statement and I will be happy to answer any questions that I can.

*Statement of*

# The American Legion

**1608 K STREET, N. W.**
**WASHINGTON, D. C. 20006**

by

PAUL S. EGAN, DEPUTY DIRECTOR
NATIONAL LEGISLATIVE COMMISSION
THE AMERICAN LEGION

and

JOHN F. SOMMER, JR., DEPUTY DIRECTOR
NATIONAL VETERANS AFFAIRS AND REHABILITATION COMMISSION
THE AMERICAN LEGION

before the

SUBCOMMITTEE ON COMPENSATION, PENSION AND INSURANCE
COMMITTEE ON VETERANS AFFAIRS
U.S. HOUSE OF REPRESENTATIVES

on

## PRESUMPTION OF SERVICE CONNECTION FOR AGENT ORANGE EXPOSURE RESIDUALS

APRIL 26, 1983

# STATEMENT OF JOHN F. SOMMER, JR., DEPUTY DIRECTOR
NATIONAL VETERANS AFFAIRS AND REHABILITATION COMMISSION
THE AMERICAN LEGION
BEFORE THE SUBCOMMITTEE ON COMPENSATION, PENSION AND INSURANCE
COMMITTEE ON VETERANS AFFAIRS
HOUSE OF REPRESENTATIVES
APRIL 26, 1983

Mr. Chairman and Members of the Subcommittee:

The American Legion is pleased to have this opportunity to appear before the Subcommittee today to present our views on legislation that would establish a presumption of service connection for certain diseases related to exposure to Agent Orange and other herbicides or environmental hazards that have been incurred by Vietnam veterans.

The American Legion policy on Agent Orange is set forth in Resolution No. 410 (Iowa), approved by the 1982 National Convention (copy attached). Our policy on compensation payable to Vietnam veterans for Agent Orange related problems, as provided in the resolution, mandates this organization to support legislation to establish a mechanism for presumption of service connection for disabilities found to be caused by Agent Orange, based on scientific and medical evidence. In other words, diseases suffered by Vietnam veterans that bear a causal relationship to Agent Orange exposure as determined by competent scientific authority should be presumed to be service-connected, regardless of how long after service they become manifest, and compensation should be paid for them based upon the degree of severity of the disabilities.

The legislative proposal currently under consideration, H.R. 1961, would, in part, amend section 312 of title 38, United States

Code - which pertains to presumptions relating to certain diseases and disabilities - by adding a new subsection. This subsection would provide that in the case of a veteran who served in Southeast Asia during the Vietnam era and who after such service suffers from soft tissue sarcomas, porphyria cutanea tarda, active and residual chloracne, or any other disease that medical research has shown may be due to exposure to herbicides, chemicals, medications, or environmental hazards or conditions, as determined by the Administrator and prescribed by regulation, the disease shall be considered to have been incurred in or aggravated by service, notwithstanding that there is no record of evidence of such disease during the period of service.

This measure also provides the Administrator with the authority to determine what other diseases may be included, based upon scientific evidence, and to promulgate regulations relating to the additional diseases, to include a specification of the standards used in making the determination.

Mr. Chairman, The American Legion does not make a practice of supporting any legislative or administrative issue because it may seem to be the popular thing to do, or due to the fact the issue may be politically attractive. The positions of this organization are based upon our understanding of an issue acquired through existing knowledge and objective research of pertinent subject matter. The Legion, since early 1978 has been involved in every aspect of the issue of Agent Orange. The effects of the herbicide on those Vietnam veterans who were exposed to it in Southeast Asia continues to be a high priority issue of the organization.

Our review has shown that there is scientific evidence that the three specific conditions contained in H.R. 1961 may result from dioxin exposure, and we would like to share the following information with the Subcommittee.

SOFT TISSUE SARCOMAS

In reviewing a Respondent's Prehearing Brief on the Risks Associated With the Registered Uses of 2,4,5-T and Silvex, prepared for the U.S. Environmental Protection Agency and dated January 25, 1980, we noted the following:

"Two recent case-control studies of the relation between cancer and occupational exposure to phenoxyacetic acids and chlorophenols present persuasive evidence that human exposure to pesticide products containing 2,4,5-T and/or dioxin constitutes a carcinogenic hazard. Each of these new studies found statistically significant increases in the risk of malignant mesenchymal tumors of the soft-tissue (soft-tissue sarcomas) related to occupational exposure to phenoxy herbicides and to occupational exposure to chlorophenols. Each study utilized an entirely separate population of cases and controls, and the studies thus clearly corroborate and replicate one another.

"In a case-control study of the risk of soft-tissue sarcomas in a population in Northern Sweden which includes forestry, sawmill, and paper pulp workers, Hardell and Sandstrom found that individuals previously exposed to phenoxyacetic acid hericides, primarily 2,4,5-T and 2,4-D, had a relative risk for soft-tissue sarcomas 5.3 times greater than unexposed individuals. They also found that individuals previously exposed to chlorophenols, which like 2,4,5-T and silvex contain chlorinated dibenzodioxins as impurities, had a relative risk for soft-tissue sarcomas 6.6 times greater than unexposed individuals. Each of these findings was highly significant in statistical terms. In both instances, p was less than .001, indicating that the observed relative risk would occur by chance alone less than one time in a thousand.

"Soft-tissue sarcomas are a rare variety of cancer. A case-control study like the one conducted by Hardell and Sandstrom is generally recognized as the most effective epidemiologic method for documenting the relationship between a particular causative factor and a rare type of tumor. The protocol used by Hardell and Sandstrom in selecting controls excluded confounding factors such as sex, age, place of residence, and year of death. In addition, the authors evaluated the possible confounding effect of factors such as occupation, smoking habits, DDT exposure, and exposure to exhaust from motorized sawing and found no evidence that any of these factors accounted for the observed increase in relative risk. Thus, it is extremely unlikely that the results obtained by Hardell and Sandstrom were materially affected by uncontrolled confounding factors or other defects in the design or execution of the study.

"In a subsequent case-control study in an entirely separate population in southern Sweden, Eriksson et. al. found that individuals previously exposed to pheynoxy herbicides had a relative risk for soft-tissue sarcomas 6.8 times greater than unexposed individuals (p less than .001). They also found that individuals previously exposed to chlorophenols had a relative risk for soft-tissue sarcomas 3.3 times greater than unexposed individuals (p less than .01). Thus, the results in the case-control study by Eriksson et. al. confirm and essentially replicate the results obtained by Hardell and Sandstrom."

The same brief contains the following information on stomach cancers among workers exposed to phenoxy herbicides:

"Studies of the incidence of disease in cohorts of exposed workers provide additional evidence that human exposure to pesticide products containing 2,4,5-T and/or TCDD constitutes a carcinogenic hazard. Two separate epidemiological studies of cohorts of workers exposed to 2,4,5-T and to TCDD have each documented a statistically significant increase in the incidence of stomach cancers among such workers.

"In a recent updating of an initially inconclusive study of mortality and tumor incidence in a cohort of Swedish railroad workers, Axelson et. al. reported a significant excess incidence of stomach cancer among those workers first exposed to phenoxy herbicides more than ten years earlier. Individuals in the Axelson cohort had previously been exposed to a number of herbicides, particularly phenoxyacetic acids (including 2,4,5-T) and amitrole. In the subcohort exposed to phenoxy herbicides but not to amitrole, the observed incidence of stomach tumors was 6.1 times greater than the expected incidence, after allowing for a ten year induction period. In the subcohort of all workers exposed to phenoxy herbicides (including those also exposed to amitrole), the observed incidence of stomach tumors was 5.9 times greater than the expected incidence, after allowing for a ten year induction period. Despite the relatively small size of each of these subcohorts, each of these findings was statistically significant. In both instances, p was less than .01, indicating that the observed excess incidence would occur by chance alone less than one time in a hundred.

"In another study of a cohort of workers exposed to TCDD during and after a runaway reaction in 1953 at a trichlorophenol plant in Ludwigshafen, Germany, Thiess and Frentzel-Beyme also reported an excess incidence of stomach cancer among exposed workers. Immediately following the Ludwigshafen incident, many of the exposed workers developed chloracne, indicating dioxin exposure.

"The observed incidence of stomach cancer in the cohort of Ludwigshafen workers who may have been exposed to TCDD during the incident or subsequent cleanup operations, was approximately five times greater then the expected incidence. (p less than .025). Moreover, further analysis indicates that the observed incidence of stomach cancer in the Ludwigshafen cohort is more than seven times greater than the expected incidence, after allowing for a ten year induction or latency period. Thus, the results obtained by Thiess and Frentzel-Beyme corroborate the findings of Axelson, indicating that exposure to TCDD or preparations containing TCDD has been clearly associated with an excess risk of stomach cancer in humans."

In reviewing other available literature it has been found that there have been a number of studies of chemical plant employees exposed to dioxin who subsequently developed soft tissue sarcomas.

Because of the small numbers of individuals involved the general feeling has been that the findings are of little significance. However, we note with interest an article by Drs. P.A. Honchar and W.E. Halperin that appeared in the January, 1981 edition of The Lancet, that discusses their review of such cases. The article states, in part, "None of the four cohorts individually resulted in a reported excess risk for soft tissue sarcoma. When combined, however, the three cases from the four cohorts suggest a common pattern. This finding supports the concept of combining small cohorts of workers with a common occupational exposure for analysis."

The Veterans Administration does not accept the results of the Swedish epidemiological case control studies because the agency feels there were insufficient numbers of exposed individuals involved in the studies to make them scientifically valid.

The American Legion feels that the evidence is adequate enough to have raised a level of suspected cause so as to be sufficient that reasonable doubt should be weighed in favor of the Vietnam veteran and service connection should be presumed for the condition.

## PORPHYRIA CUTANEA TARDA AND CHLORACNE

Porphyria cutanea tarda results from a disturbance in the capacity to break down hemoglobin, which leads to high levels of porphyrins (nitrogen-containing organic compounds) in the liver and urine. In many, if not most cases there is also skin involvement.

In reviewing available literature we have found a substantial number of references to the connection between dioxins and porphyria cutanea tarda. Many references to PCT are made in conjunction with those pertaining to chloracne, so we have combined our views and comments on the two conditions.

A recent report by the Council on Scientific Affairs of the American Medical Association discussing the health effects of Agent Orange and dioxin contaminants states in part, "Two of the more pronounced biological effects of some of the chlorinated dioxins are their tendency to cause chloracne (especially in the rabbit, nude mouse, monkey and man) and the accumulation of fluid (ascites) in the pericardium and peritoneal cavity of chicks." The report further states, " Other acute toxic reactions to dioxin include liver and renal damage, porphyria cutanea tarda, hyperpigmentation, hirsutism, ..."

An Environmental Protection Agency report on dioxins, in the chapter relating to health effects notes, "Although chloracne is a common indicator of 2,3,7,8-TCDD exposure in humans and some animals, chronic exposure to this dioxin can affect many organ systems. In addition to chloracne, another dermatologic manifestation of exposure is porphyria cutanea tarda (PCT), a photosensitive dermatosis caused by altered porphyrin metabolism." The following is also mentioned in the report: "Dermatologic diseases are perhaps the most sensitive indicators of 2,3,7,8-TCDD exposure and toxicity in humans. Although chloracne is the most frequently observed dermatosis, PCT has been observed in as many as 10 percent of a group of occupationally exposed workers (Purkyne et. al. 1974)."

A VA Chloracne Task Force report dated November 30, 1982 contains the following: "The examination will incorporate new findings and old confirmed by the 3rd International Symposium on Chlorinated Dioxin and Related Compounds in Salzburg, Austria, October 12-14, 1982. New and confirmed findings are:

1) Chloracne may persist in 25-50% of cases, up to 30 years, as shown by the longest follow-up to date of an industrial accident. It was previously believed it cleared in a few years.

2) Porphyria cutanea tarda may result from low chronic exposure to dioxin (as may have occurred in some Vietnam veterans). Therefore, screening of urine, stool and any liver biopsy tissue will be done for porphyrins.

3) Hirsutism of face and hyperpigmentation may be due to PCT."

In an October 1982 report by a Subcommittee of the National Academy of Sciences on a review of the protocol for the VA Agent Orange study, it was recommended, in part, that the protocols should be focused, if possible, on known effects on humans and animals of exposure to phenoxy herbicides contaminated with TCDD (dioxin). Included among the conditions were effects on the skin and liver function, including porphyrin metabolism.

Mr. Chairman, these are but a few of the references to the relationship between dioxin exposure, and porphyria cutanea tarda and chloracne that we uncovered during our review. However, the Legion feels that these representative examples are sufficient to illustrate that a causal relationship does in fact exist, and that Vietnam veterans exposed to dioxin while serving in Vietnam and who incur these disabilities, should be presumed to be service-connected and compensation awarded where applicable.

With respect to the diseases that would be listed in regulations prescribed by the Administrator, as required by this measure, The American Legion has urged the Centers for Disease Control to release all relevant findings as they become available during the epidemiological study of the long-term health effects of Agent Orange exposure, mandated by PL 96-151. And while we are on this subject, it must be said that we are encouraged by the manner in which CDC has performed since accepting the responsibility for the Agent Orange study in January. Within a matter of days following the signing of the Interagency Agreement with the Veterans Administration, CDC officials met with The American Legion, other veterans organizations and representatives of other government agencies involved in the study, to discuss the study and elicit input and recommendations relating to the research. The CDC officials have assured that they will maintain open lines of communication throughout the study.

In turn, we have offered the cooperation of the Legion in encouraging the participation of Vietnam veterans in the study, as we are aware of the importance that such participation will play in the success of the Agent Orange study.

Mr. Chairman, we understand and appreciate that the intent of this legislation must undergo the prudent deliberation of this Subcommittee, and, as with other issues where differences of opinion in the scientific community exist, we have no doubt that H.R. 1961 will be closely but objectively scrutinized. May we point out that other presumptions have been established by Congress based upon a lesser amount of evidence than is currently available in support of the instant legislation.

The American Legion thanks you Mr. Chairman for your timeliness in scheduling this hearing on an issue that is of great importance to Vietnam veterans; and, again, we appreciate this opportunity to provide the Subcommittee with the views of this organization.

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TESTIMONY OF LEWIS M. MILFORD
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BEFORE THE
SUBCOMMITTEE ON COMPENSATION AND PENSIONS
COMMITTEE ON VETERANS AFFAIRS

UNITED STATES HOUSE OF REPRESENTATIVES

APRIL 26, 1983

The American University Washington College of Law

MR. CHAIRMAN AND MEMBERS OF THE SUBCOMMITTEE:

My name is Lewis M. Milford. I am a lawyer with the National Veterans Law Center in Washington, D.C. I am also on the faculty of the Washington College of Law, American University, where I direct a law clinic and teach a seminar in toxic substances litigation. The Center is a public interest law firm which represents veterans in federal court litigation and in federal administrative hearings. The Center has been involved in the controversy over Agent Orange since 1978.

Congressman Daschle's introduction of the Vietnam Veterans Agent Orange Relief Act (H.R. 1961) should be welcomed by those concerned with fair treatment of Vietnam veterans. The bill would compensate certain veterans suffering from the effects of Agent Orange exposure in Vietnam. It is a limited measure, requiring compensation only for three diseases definitely linked in the scientific literature to the health consequences of dioxin exposure. The strong support of the veterans organizations expressed today shows that this is a measure whose time finally has come.

The bill should be supported for three compelling reasons. First, the scientific evidence clearly shows that the three diseases, soft tissue sarcoma, a liver condition known as porphyria cutenea tarda and the skin condition known as chloracne are all associated wich dioxin exposure. Public health agencies of the federal government have strongly confirmed the scientific connection for the most serious disease of the three, soft tissue sarcoma. Second, it is now time for the government to make this policy because of this compelling evidence and because the Veterans Administration (VA) has demonstrated that it will continue to ignore valid scientific evidence in making compensation decisions on veterans' Agent Orange claims. Third, this legislative proposal is consistent with existing legislative and administrative presumptions to award service connected benefits on the basis of a similar body of scientific evidence.

## SCIENTIFIC BASIS FOR PRESUMPTIONS

The scientific support for this measure will be more explained by scientific experts during tomorrow's hearings. Our testimony is not, therefore, intended to survey the existing literature. However, we want to point out that the federal government's own public health experts have offered the best evidence for the causal connection between dioxin and ill health effects.

The most important health studies on dioxin's cancer connection are from Europe. The causal relationship between dioxin exposure and a rare form of cancer, soft tissue sarcoma, has been recognized by the Environmental Protection Agency (EPA), the Centers for Disease Control (CDC) and the National Institutes for Occupational Safety and Health (NIOSH). In 1980, the EPA relied on these studies to argue for the emergency suspension of 2,45-T in domestic use. In its pre-hearing brief, EPA stated that there was a "growing body" of epidemiological evidence that established a "clear association" between exposure to 2,45-T and dioxin and cancer in humans. EPA's lawyer said that the latest studies by Hardell and Sandstrom show "statistically significant increases in the risk of malignant mesenchymal tumors of the soft tissue (soft tissue sarcomas) related to occupational exposure to phenoxy herbicides and to occupational exposure to chlorophenols."1/

A year later in 1981, NIOSH researchers concluded that, "[C]ase-control studies from Sweden demonstrate relative risks of 6·2 and 5·1 for occupational exposure to phenoxy herbicides and chlorophenols among cases of soft-tissue sarcoma."2/ These studies led the researchers to undertake a study of American workers potentially exposed to phenoxy herbicides. Finally, the federal government's top scientist working on Agent Orange wrote in 1980 that the European soft-tissue sarcoma "studies show a correlation between exposure to phenoxy herbicides and increased risk of some form of cancer."3/

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1/ Pesticide and Chemical News, Feb. 13, 1980.

2/ P.A. Honchar and W.E. Halperin, 2,45-T and Soft Tissue Sarcoma, Lancet (Jan. 31, 1981).

3/ John Moore, D.V.M., Evaluation of Five Scientific Papers on the Carcinogenicity of Chemicals That Were Constituents of Agent Orange (Memorandum to Scientific Panel) (June 25, 1980).

These human studies demonstrate a strong statistical correlation between dioxin exposure and soft tissue sarcoma. This is not a frivolous problem; at least three Vietnam veterans exposed to Agent Orange have been identified as having soft tissue sarcoma.[4] There is little doubt that legal causation is established where a valid epidemiological study demonstrates a statistically significant relationship between exposure to a toxic substance and a certain disease. Causal associations from epidemiological studies are the only means science has to connect disease to environmental exposures. And in toxic substances personal injury litigation, such a statistical correlation is virtually the functional equivalent of causation.[5]

We believe that these epidemiological studies would demonstrate to any court of law that a veteran's soft tissue sarcoma was caused by dioxin exposure. We are convinced that a veteran's burden of proof on causation would be met by submission of these scientific studies. Therefore, we are equally convinced that the studies are a sound basis for a legislative presumption for service connection. The VA certainly should not require more scientific proof than would be necessary to prove causation in a court of law.

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[4] P. Ravi Sarma and Julian Jacobs, Thoracic Soft-Tissue Sarcoma in Vietnam Veterans Exposed to Agent Orange, New England Journal of Medicine (May 6, 1982).

[5] See generally, Harley, Proof of Causation in Environmental Litigation, in Tort Actions for Cancer and Lung Disease Due to Environmental Pollution (1977).

The same argument applies to chloracne and porphyria cutanea tarda. It is beyond dispute that chloracne is caused by dioxin exposure. And the liver disease also has been associated in humans with dioxin exposure, in particular from the accident at the Monsanto plant in Nitro, West Virginia.

## FEDERAL GOVERNMENT POSITION

For several years, the VA has taken a familiar position on the issue of dioxin causation for health problems in Vietnam veterans. The agency has refused to identify how much scientific evidence would establish a causal connection. No standard of proof in a VA Agent Orange claim has ever been suggested by the agency. At the same time, the VA says that existing scientific data on dioxin, the European studies, are not sufficient to demonstrate causation. The bill would put a stop to this deception.

In 1978, the VA issued a Program Guide to its claim offices that concluded that only chloracne could be associated with dioxin exposure. Litigation over that policy was filed to require the agency to give veterans and the public an opportunity to share their views with the agency on this important issue.6/ The VA refused to allow for such public participation in the development of causation standards for Agent Orange claims. The agency continues to resist, three years later, any efforts to have its Agent Orange compensation

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6/ White v. Nimmo, No. 79-1426 (D.D.C. filed May 31, 1979).

policy subjected to public scrutiny. As a result, there are no agency standards to inform veterans about which scientific evidence is considered relevant on the issue of causation. Veterans are left completely in the dark about how to prepare a claim based on existing evidence. No agency policy on compensation policy exists other than the five year old Program Guide.

In the most recent VA statement on Agent Orange, the VA suggests that the European studies show a relationship between dioxin and cancer, but that this is not enough to prove causation. The agency stated, "[S]everal epidemiological studies conducted by Swedish scientists have reported evidence of a relationship between soft-tissue sarcoma and exposure to phenoxy herbicides and dioxin."[7/] Having said that, the report goes on to argue that other studies in Finland and Australia do not show such a relationship. What is at work here is extremely disturbing and is of great importance to this issue. The VA appears to be arguing that one, two or even five separate human health studies on dioxin are not enough for the agency to conclude that dioxin causes soft tissue sarcomas. If this is the government's stance, it represents a dramatic reversal in public health policy from

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[7/] Agent Orange Research Update in Agent Orange Review, Veterans Administration, Vol. 2, No. 1 (April 1983).

the position of the last Administration.

The effort by VA to increase the burden of proof for veterans on the dangers of dioxin is made dangerously clear by the recent statements of Dr. Al Young, the Air Force officer who is now the VA's principal scientific adviser on Agent Orange. In a televised interview, he said the following:

> I think the most important thing to remember is that the studies simply aren't in yet. Where the Swedes found soft-tissue sarcomas, the Finns did not. The New Zealand scientists did not find soft-tissue sarcomas.[8]

The agency is playing a dangerous game of ignoring human health evidence with which it disagrees. It is a deceptive argument that ignores a long history of public health policy based on animal and epidemiological data, and it conflicts with legal standards of proof well established in personal injury actions involving toxic substances. Even more importantly, it is a position witn no limits and no principle. There will always be some conflict in scientific evidence. But our society has made a judgment to regulate dangerous chemicals and compensate for damage from exposure to them based on the available scientific evidence that allows for a fair and compassionate judgment. We have made a social decision to make public policy in a way that favors the award of compensation for probable damage. We have decided that it is better to resolve scientific uncertainty to relieve individual harm, rather than wait for the ultimate and "conclusive" proof in the form of dead bodies.

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[8] Dr. Alvin Young, How Dangerous is Dioxin?, ABC Nightline Transcript at 7 (March 15, 1983).

The inter-agency Agent Orange Working Group, less visible recently, made an important recommendation a few years ago. It recognized that no scientific study on Agent Orange would ever produce a "conclusive" answer. Rather, it said that compensation is fundamentally one of "broad social policy that both the administration and Congress must confront." The VA would prefer to avoid the policy debate because that raises questions about the fairness and justice of continuing to deny veterans compensation as the evidence mounts. And any policy discussion must begin with questions that go behind the VA's repeated statement that no "conclusive" evidence exists. That remains a false standard, a delaying tactic, until an answer is given about what the current studies mean and why they are insufficient to make compensation policy.

This proposal legislation takes up the challenge of the Working Group to confront important social policy that is the Agent Orange issue. Congress must act where the VA has refused to act.

## TIMES BEACH

The federal government buy-out of dioxin-contaminated Times Beach shows what can happen when science is used in support of good public health policy. The Missouri case illustrates two things about the federal government and Agent Orange. First, the case demonstrates how serious the dangers of dioxin are viewed even by the public health agencies operating under this Administration. Second, the buy-out demonstrates how political pressure converts interminable scientific debates into a manageable political solution.

Also, the federal government spent millions of dollars to purchase an entire community because of the present risk of disease. The expenditure was not based on proof of any current health problems, a compassionate approach that stands in marked contrast to the attitude of the VA with regard to the same dangerous chemical. Since the Times Beach affair came to public attention, the VA has been busy devising scientific distinctions between that devastated community and the larger community of Vietnam veterans exposed to Agent Orange. In my opinion, the problem is that the VA does not really believe Agent Orange is a public health problem. We still see VA officials on national television blaming the victims for the problem, attributing their concerns to the "tremendous amount of emotion associated with dioxin. . . when you can't see it, there's a great deal of fear."[9/] This kind of talk is on the same level as those who used to make claims about the safety of dioxin by promising to drink it. Again, Congress has been forced to act in the face of these cavalier attitudes.

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[9/] Dr. Al Young, Nightline, supra at 8.

## EVIDENCE TO SUPPORT A SIMILAR VA PRESUMPTION

The VA appears to acknowledge that the European studies show a significant increase in sarcomas in workers exposed to dioxin. However, the agency does not consider this sufficient evidence of causation, if not for those workers, at least for Vietnam veterans. However, the agency has found that a statistical increase in heart disease among veteran amputees was sufficient evidence of causation, even though the underlying scientific studies expressly stated that there was no evidence on the cause of the disease.

In P.L. 94-433, Congress mandated the VA Administrator to conduct a scientific study to determine if there is a causal relationship between the amputation of an extremity and the subsequent development of cardiovascular disorders. The study was made by the Medical Follow-up Agency of the National Academy of Sciences. The study concluded that veterans who suffered an amputation of both legs or of one leg at or above the knee have a significantly higher risk of dying from diseases of the cardiovascular disease.

However, the study could not determine the cause of the increased risk of cardiovascular disease. The report concluded that, "[T]he study was not designed to test specific hypotheses about the causes of the increased risk."[10] The VA then issued a regulation to award service connection for heart disease that developed in veterans with such amputations.

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[10] Report on Service Connected Traumatic Limb Amputations and Subsequent Mortality from Cardiovascular Disease and Other Causes of Death, National Academy of Sciences (Dec. 11, 1978).

The point of this comparison is to show that the VA has not required "conclusive" causal evidence to award benefits in other cases. Statistically significant evidence of an increased risk of disease has been sufficient in the heart disease area. The study evidence was based on veterans but that is not controlling. Statistical evidence from human studies should be sufficient to compensate any other similarly exposed and injured group of persons.

TESTIMONY OF LT. COL. DAVID J. PASSAMANECK, USA, RET.
NATIONAL LEGISLATIVE DIRECTOR

Before The

SUBCOMMITTEE ON COMPENSATION, PENSION & INSURANCE

Of The

COMMITTEE ON VETERANS' AFFAIRS

U. S. HOUSE OF REPRESENTATIVES

April 26, 1983

AMVETS appreciates the opportunity to present our views concerning the highly sensitive issue of the infectious consequenses of the exposure of our service personnel in Southeast Asia, during the period of the Vietnam War to phenoxy herbicides, including so-called "Agent Orange.

Since the issue was raised by concerned Vietnam veterans approximately 15 years ago, we have witnessed a continuous monumental exercise in the generation of documents reflecting the collective lack of definitive medical and scientific information regarding the epidemiological connection between exposure to Agent Orange and various physical disabilities, including, cancer, skin lesions, birth defects, liver disease and even psychological problems. This documentary mountain has been generously contributed to by the Veterans Administration, the Congress, other government agencies, and many private citizen groups, including, of course, veterans organizations.

Two substantive legislative enactments have resulted from the widespread concern about the subject. Public Law 96-151, signed on December 20, 1979, directed the VA to conduct an epidemiological study of persons who were exposed to phenoxy herbicides and to determine the medical consequences, if any of that exposure. Interagency contracts have been signed between the VA and the Center for Disease Control (CDC) and expenditure of approximately $3,000,000 has been approved by the Office of Management and Budget to conduct the study, which, optimistically speaking should result in a first report to the Congress sometime in 1988 or 1989. In addition, Public Law 97-72 offers Vietnam veterans who request it, priority physical examinations and diagnostic studies which could result in prolonged treatment for those conditions medically determined to be caused by Agent Orange. AMVETS believes that the authority for the diagnostic treatment in Public Law 97-72 already existed in Title 38 in connection with the routine adjudication of claims for compensation for any service-connected disability. We are appreciative, however, of the motivational thrust of the full Committee in positively highlighting the problem in Public Law 97-72.

Without reiterating the sad and often confusing story of delay and inaction by the VA in the conduct of the epidemiological study mandated by Public Law 96-151, we would simply endorse the remarks of the former Administrator of Veterans Affairs, Mr. Nimmo, in August of 1982, when he said, "I found our research stuck on dead center. People had good intentions, but nothing was happening.' We are sorry to say that Mr. Nimmo's remarks are still applicable at the present time.

AMVETS views the purpose of H.R. 1961 as building a much needed fire under the VA to spur completion of the determinative studies relating to Agent Orange with a view toward ending the terrible uncertainty and anxiety afflicting our Vietnam veterans and their families, and compensating those who may be found entitled to service-connected benefits as the result of exposure to this lingering hazard of their service.

We believe, however, that the responsibility for creating epidemiologic presumptions in Title 38 belongs to the Congress and should not be shifted to the VA or any other executive agency. It is one thing to build a fire under the VA to hopefully insure compliance with Public Law 96-151 before all of our affected Vietnam veterans become too old and sick to take advantage of it. It is quite another thing to require the Administrator to gather the firewood and supply the matches. AMVETS, therefore, supports enactment of H.R. 1961 with the exception of that portion of Section 3 which adds subsection (d)(2)(A)(IV) and (B) to Section 312 of Title 38 United States Codes.

This concludes my testimony. I am prepared to answer any question you may have.

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STATEMENT OF
JOHN F. TERZANO, LEGISLATIVE DIRECTOR
VIETNAM VETERANS OF AMERICA
accompanied by
LESLIE A. PLATT, SPECIAL COUNSEL
VIETNAM VETERANS OF AMERICA
before the
SUBCOMMITTEE ON COMPENSATION, PENSION, AND INSURANCE
OF THE
HOUSE VETERANS AFFAIRS COMMITTEE

APRIL 26, 1983

Mr. Chairman and Members of the Subcommittee, I am John Terzano, Legislative Director of the Vietnam Veterans of America. Accompanying me today is Leslie Platt, VVA Special Counsel. Mr. Chairman, I am honored to be here today to appear before the Subcommittee on Compensation, Pension and Insurance to state the views of the Vietnam Veterans of America regarding H.R. 1961, the Vietnam Veterans Agent Orange Relief Act. My testimony today will be brief and to the point.

Four years ago, the Vietnam Veterans of America was the first witness at the first Congressional hearing on Agent Orange and the possible adverse health effects it may have caused to those who served in the U.S. Armed Forces in Vietnam. In those hearings, VVA called for the establishment of a presumption of service connection for cancer and liver disorders experienced by America's Vietnam veterans.

Those hearings in 1979 culminated in the introduction in the Congress of the first bill specifically focused on the problem of Agent Orange and the health status of America's Vietnam veterans and their families. That bill was introduced by Congressmen Thomas Daschle and David Bonior, who were then, and remain today, pioneers and leaders in the fight for basic equity for those who served their country in Vietnam.

Much has happened since those first hearings. Perhaps most important, offical Washington at both ends of Pennsylvania Avenue now recognizes Vietnam veterans may already be, or may in the future become, ill as a result of their service in Vietnam. This recognition was slow in coming, and much yet remains to be done.

However, we are pleased about the progress which the Federal Government has made. The Government has acknowledged its responsibility to conduct a comprehensive research program to learn more about both the possible connection between exposure to phenoxy herbicides in Vietnam and health decrements, and more broadly about the possibility that many veterans may now be suffering, or may run increased future risk of suffering, serious adverse health effects as a result of something in the Vietnam experience. Overall, we clearly now have the framework and basic elements of a concerted, responsible Government research program.

VVA has in the past pointed out--and it will in the future continue to comment on--areas where we believe there is lethargy or inadequate attention or resources to keep the research moving forward.

Also, the Veterans Administration has completed a creditable worldwide scientific literature review and analysis. And, most importantly, veterans who may be suffering adverse health effects as a result of their exposure to herbicides in Vietnam are now entitled by law to receive a priority on health care from the VA.

These forward steps did not come easily, but were won only after hard work and dedication by many individuals and institutions. This Committee has been and remains a critical factor in the formation of responsible social policy on this issue, and we remain confident that the Committee will continue to exercise leadership in this area.

For those of us who have been at the forefront of this fight for official recognition of, and attention to, our concerns: for those of us who were ridiculed, scorned and, in some cases thought to be just plain "off our rockers" in the early stages: for those of us who have now seen our views recognized in Federal law and multi-million dollar Government research budgets: for us, the progress of the recent past is only the beginning and much remains undone. Our concerns are now shared by millions of Americans who are becoming acutely aware of the potential hazards of these chemicals.

We are still years away from being likely to have definitive information on the relative health status of ground troops who served in Vietnam as compared to their peers who did not serve. And, for many of us, the knowledge of whether we can safely have children or are likely to die prematurely of cancer or suffer other serious maladies as a Vietnam legacy is still the most important, ultimate issue.

However, we also seek basic equity for Vietnam veterans while we wait for the scientists to finish their end of the job. That is why VVA is here today, approximately four years after those first Congressional hearings, and five years after Agent Orange first surfaced as a national issue, again presenting testimony on behalf of America's Vietnam veterans on the same core issue--presumption of service connection for disability compensation for serious health problems which the scientific literature clearly indicated may be attributable to exposure to phenoxy herbicides.

Mr. Chairman, I am not here today to detail the sound scientific basis for H.R. 1961. There is no doubt that the time has long-since come for the Congress to provide such presumptions. In a different world, the Veterans Administration itself might well have done so under its administrative authority, but, as with priority health care for Vietnam veterans, Congressional directions by statute were necessary.

We ask several questions about the issue of compensation. How can the same Federal Government that buys out Times Beach, Missouri, for over $30 million continue to turn its back on a group of individuals who have given so much of themselves for their country? What kind and how much scientific data will be enough? When will the Congress, the Executive Branch, and the Veterans Administration acknowledge and deal responsibly with the existing data which were deemed sufficient for addressing Times Beach but are somehow found deficient for America's veterans?

Also, after years of denying chloracne claims, the VA now insists that it pays those claims. What was that one piece of additional evidence that finally tipped the scales of justice towards the veterans? And what piece of evidence will convince the VA again to tip the scales in favor of the veteran?

Mr. Chairman, H.R. 1961 is limited legislation. It is moderate and carefully circumscribed. If is becomes law, it will only help a small number of sick veterans. Yet, VVA believes it is an important step, as it will finally establish a statutory framework for dealing responsibly with the compensation issue by placing the Government on the side of the veteran.

VVA submits that there is now enough evidence on which the Congress can and should act. We submit that the Congress need only look to the VA's own scientific literature review, and EPA's recent actions, to see that evidence.

Yet, there are those who say that the evidence is not enough. There are those who feel that we still need to wait.

It has been said on numerous occasions that we cannot afford to become complacent because a comprehensive research agenda is being carried out. Even the best effort of which scientists are capable may not ultimately provide definitive, incontrovertible scientific information about the health effects of phenoxy herbicides. Joan Z. Bernstein, the Chair of the original Interagency Work Group on Phenoxy Herbicides stated in testimony over three years ago that "...In short, we may be left, after the research is done, with many of the same social policy issues we face today."

Mr. Chairman, the time has come for Congress to have the courage to begin to confront the compensation issue. H.R. 1961 is a good bill and it should be passed now. VVA strongly supports this legislation. The care and effort that Mr. Daschle and his staff and many other Members have put into developing this legislation is clearly evident.

H.R. 1961 is a bare minimum that the Government can and should do now. In fact, the bill, in my opinion, does not go far enough. Volume I, Chapter 5, of the VA's literature review lists a number of diseases for which there is ample scientific evidence to support presumptive compensation eligibility for additional disabilities.

Specifically, the VVA would strongly recommend making the following changes to the bill:

* A broader liver disorder presumption to include, but not be limited to, chronic hepatitis and othe liver disorders whose etiology is other than congenital or alcohol related. Not only is there evidence in the literature review to support this, but just last week the New York Times reported that 112 out of 130 residents of Imperial, Missouri whose blood and urine were tested by the Centers for Disease Control were told that they had abnormalities in their samples.

* A broader skin disorder presumption. Thousands of veterans have complained to the VA about various skin problems. While the VA claims that only a few are chloracne and that it does treat and compensate them, it does nothing for the overwhelming majority of those who have other skin problems.

* A presumption of peripheral neuritis, a loss of sensory function or numbness.

* A presumption for lymphomas.

In closing, Mr. Chairman, the Agent Orange story is one in which the basic issue is the government's integrity and credibility. This Committee has the opportunity to strengthen that integrity and credibility by moving forward with this compensation legislation. Then, when the final chapter is written on treatment of Vietnam veterans, this Committee will not be remembered among those who told America's Vietnam veterans that they, and the sacrifices they made over a decade ago, have been forgotten. The Committee's action on this legislation will be noted and remembered.

Please do not fail to stand by us and this responsible, moderate legislation. The time has come to move forward.

Thank you.

# TESTIMONY OF CONGRESSWOMAN BARBARA BOXER
# BEFORE THE VETERANS' AFFAIRS SUBCOMMITTEE
# ON COMPENSATION, PENSION AND INSURANCE

APRIL 27, 1983

A recent song about Vietnam by Billy Joel says, in part:

> We came in spastic -
> We left in plastic -
> As numbered corpses

For the many of those who didn't come home in a box --

For the many of those whose names are not listed on the Vietnam Memorial -- life has become a tortured and difficult existence. And, the Veterans Administration's painfully slow decision-making on the health affects of Agent Orange is making it more tortured and more difficult.

Despite numerous respected studies linking the diseases in H.R. 1961 with Agent Orange, there is still not automatic compensation to Vietman Veterans suffering from certain cancers and skin ailments.

What can these Vietnam Veterans do to make the federal government listen? Under the Feres doctrine, emanating from a Supreme Court decision, they cannot sue the federal government for service related ailments although their exposure to Agent Orange is unquestioned. And, the decision to use Agent Orange was made by the U.S. government. Lawsuits against the chemical companies which produce Agent Orange are a slow and arduous process.

The answer is that we, in Congress, are their only hope. They can't sue. The Veterans Administration hasn't been moved despite the studies. The veterans in my district tell me that since the Veterans Administration rejects Agent Orange claims, as a matter of course, many veterans don't even bother consulting the Veterans Administration.

Mr. Chairman, here are three very brief histories of veterans in my district. Due to the abbreviated time I have before you, these capsule histories do not even begin to convey the scope of the suffering endured as a result of exposure to Agent Orange and other herbicides used in South East Asia.

Case No. 1

Veteran in Vietnam 1967 to 1969 as an Army Infantry medic. He saw helicopters spraying defoilants and he camped in and travelled through defoliated areas, drinking the water from the ground and rivers. He has suffered from (partial list) a brain tumor, which was operated on, skin rashes and spots, liver disease, stomach disorders, headaches, dizziness, insomnia, depression, rages, decreased smell and taste, stiff and numb joints and extremities. He has a son born with birth defects (deformed toes on both feet) who has difficulty walking, and who gets sick often. Veteran applied for V.A. benefits, and received a letter from the V.A. conceding his exposure to herbicides in the Service but denying compensation.

Case No. 2

Veteran in Vietnam 1968 to 1969 serving as Marine Corps Infantry Squad Leader. He saw defoliants being sprayed, passed through defoliated brush, and walked, slept, drank from and bathed in defoliated areas. He has suffered from testicular cancer, which required surgery, and which has resulted in impotency and sterility; other medical problems include (partial list) chest pains, night sweats, abdominal cramps, skin rashes with blisters, stiffness, pain, numbness in extremities, and personality changes. He filed a V.A. compensation claim which was denied.

Case No. 3

Died at age 35 of carcinoma of the lungs. He was in the Marine Corps for ten years and served in Vietnam from March to September 1968. He slept, worked and walked through defoliated areas sprayed with herbicides. The V.A. denied any claim that his illness was a result of Agent Orange exposure. He left a widow and three children aged 3, 5 and 11.

Mr. Chairman, we don't know how to undo the physical damage caused by Agent Orange, but we will try. We don't know how to undo the psychological trauma of the Vietnam War, but we will try. But, what we can and must do now is compensate all of those who have been wounded in our wars.

If someone lost an arm or a leg in service to our country, he or she was rightly compensated. If someone lost sight or hearing, he or she was rightly compensated.

The chemicals in Agent Orange inflict a different kind of damage -- slower, and sometimes more subtle damage -- but, without question, damage, sometimes fatal damage. This damage must be compensated. We have the studies. We have been patient.

The Congress sent men and women to Vietnam to fight. Now, the Congress must be ready to pick up the pieces. And, the pieces are there in my California district, and all over this nation, where several thousand ~~hundred~~ men and women continue to seek help.

I commend Mr. Daschle for authoring H.R. 1961. I am proud to be supporting this bill.

# Testimony on Agent Orange and H.R. 1961 by Samuel S. Epstein, M.D.

School of Public Health
University of Illinois Medical Center
Chicago, Illinois

before the House Veteran's Affairs
Subcommittee on Compensation and Pensions

April 27, 1983

## A. Introduction

Mr. Daschle, members of the House Subcommittee on Compensation and Pensions, my name is Samuel Epstein, M.D., and I am professor of occupational and environmental medicine in the Department of Preventative Medicine at the University of Illinois Medical Center, Chicago.

As a human and experimental pathologist and toxicologist, I have for some three decades studied the hazardous effects of chemicals and chemical pollutants, including pesticides, herbicides, industrial chemicals, drugs, and food additives, in air, water, food and the workplace, with particular reference to delayed or chronic toxic effects, notably cancer, reproductive and genetic effects and have over two hundred and fifty scientific publications and six books in these areas. Furthermore, over the past decade, I have had increasing involvement in the interface between science and public policy, as exemplified by membership of a wide range of Federal advisory and expert committees, and by consultantships to Congress, including the Senate Committee on Public Works. Additionally, I have served over this period on the Environmental Health Advisory Committee of EPA, and more recently on its subcommittee on pesticide tolerances. Other committees on which I have also served include the 1973 Advisory Committee to the Department of Labor on Standard Setting for Occupational Carcinogens.

Of interest in the present connection is the fact that I chaired the Panels on H.E.W. Secretary Finch's 1969 Commission on Pesticides and Their Relationship to Environmental Health, which warned of the critical need to restrict useage of 2,4,5-T and 2,4-D to prevent risks of human exposure. On the basis of these recommendations, and the April 1970 hearings of Senator Hart's Subcommittee on Energy, Natural Resources and the Environment, in which I testified, H.E.W. suspended most domestic uses of 2,4,5-T. More recently, I had the privilege of testifying on H.R.6377 before the Subcommittee on Medical Facilities and Benefits in 1980.

I should like to offer Mr. Daschle, Mr. Applegate, and other subcommittee members my congratulations for their proposed legislation, H.R. 1961 which attempts to address major inequities in compensation of Agent Orange disease.

## B. Problems in Demonstration of Causality for Agent Orange Disease

The level of scientific proof generally attainable and required for the demonstration of causality is conventionally defined in terms of presumption of probabilities, rather than more absolutely. However, for Agent Orange disease not only is a high or absolute level of proof inappropriately demanded by the Veterans Administration (VA), but also the burden of proof is shifted to the veteran.

The demonstration of causality in disease due to toxic exposures requires evidence of exposure and of adverse effects, and evidence of positive associations between exposure and effect, as derived from toxicological and/or epidemiological studies. Neither scientifically or legally is evidence on causality invalidated by the possibility of unrelated contributory exposures. Nor can or should such evidence be challenged by inability to retrospectively quantitate exposure data.

The application over a decade, ending in May 1970, of multimillion gallons of concentrated 2,4,5-T formulations, apart from 2,4-D and other non-phenoxy herbicides, coupled with its heavy contamination with the highly persistent dioxin, creates the strong presumption of exposure for all military personnel in Vietnam, other than possibly those in defined groups permanently stationed in such urban centers as Saigon.

## C. Evidence for Adverse Health Effects Induced by the Dioxin Contaminant of Agent Orange

### 1. In Experimental Animals

The dioxin contaminant of Agent Orange is the most toxic synthetic chemical compound known. Dioxin is also the most potent known carcinogen and teratogen which, in relatively small groups of laboratory animals, induces cancers and injures or kills embryos at doses as low as one billionth of a gram/kg body weight/day (0.001 ug/kg).

The acute oral $LD_{50}$ of dioxin in guinea pigs and rats is about 1 and 30 ug/kg, respectively. Death following a single oral dose in rats may be delayed for weeks and appears to be due to acute liver necrosis (Poland & Kende, 1976). Mortality patterns are similar in rats and guinea pigs irrespective whether dioxin is given as single or divided doses over a 5 weeks period, indicating cumulative toxicity (Harris et al., 1973). A variety of acute toxic effects are seen at much lower doses than those required to induce mortality. For instance, a single oral 0.1 ug/kg dose induces disturbances of lipid metabolism and liver enlargement in rats (Cunningham & Williams, 1972); low doses also induce profound changes in thyroid function, as evidenced by increased $I^{131}$ uptake and increased levels of serum TSH (Bastomsky, 1977). In rodents, reduction of thymic weight (Harris et al., 1973) and, to a lesser extent, of body weight are sensitive indicators of subtoxic dioxin effects, whose characteristics tend otherwise to be both dose and species dependent. For instance, mice and rats are resistant to the acneigenic effects of dioxin, while rabbits are sensitive. Still more sensitive are monkeys, in whom chloracne and alopecia are induced by a single 400 ug/kg injection (Van Miller et al., 1976). Suggestions have been made that the high toxicity of TCDD is somehow correlated with its very marked potency in inducing microsomal liver enzymes, such as Aryl Hydrocarbon Hydroxylase (Poland & Glover, 1974).

Subacute and chronic effects of dioxin in rodents are varied and include liver necrosis and thymic atrophy (Gupta et al., 1973). Suppression of cell mediated immunity and other immunological abnormalities, including increased sensitivity to bacterial infection and reduction in delayed type hypersensitivity responses, have been consistently induced in rodents at doses of dioxin producing no other overt clinical or pathological effects, 1.0 ug/kg week or lower (Vos & Moore, 1974; Thigpen et al., 1975). Doses of 500 ppt fed over months to monkeys produced chloracne, pancytopenia, reproductive abnormalities and death (Allen et al., 1977; Allen & Van Miller, 1978). Chronic feeding of rats with 0.1 ug/kg (about 2000 pppt in diet) induced profound cardiovascular changes, including periarteritis, intravascular thrombosis, and myocardial degeneration (Kociba et al., 1978).

Dioxin induces a wide range of adverse reproductive abnormalities. Gonadotoxic effects, including testicular atrophy and necrosis and inhibition of spermatocyte development, have been recognized in rodents, chickens, and monkeys (Norback & Allen, 1973; McConnell et al., 1978).

Fetotoxic effects have been induced in every species at the lowest dose tested, including mice and rats, and monkeys at 0.001

and 0.002 ug/kg day, respectively (for review, see EPA, 1980). Administration of dioxin to pregnant rats induces fetotoxic effects, including reduced fetal weight, subcutaneous edema and intestinal hemorrhage (Sparschu et al., 1971). Various studies have demonstrated that TCDD induces teratogenic effects in mice and rats, particularly cleft palate and kidney defects, and in all other species tested (for review, see EPA, 1980); the teratogenic effects are synergized by concomitant administration of 2,4,5-T (Sparschu, et al., 1971). The lowest dose of dioxin tested in female monkeys, 0.002 ug/kg (50 ppt in diet) produced profound reproductive toxicity, including stillbirths, abortions, reduced fertility, menstrual irregularities and hormonal disturbances (EPA, 19

TCDD is the most potent known carcinogen. In tests with small groups of rats over a dose range from 1 ppt (0.0003 ug/kg) to 1000 ppb (500 ug/kg), all animals receiving doses in excess of 500 ppt died within 95 weeks. Dioxin induced a wide range of malignant tumors in doses as low as 5 ppt (Van Miller et al., 1977). These include squamous carcinomas of the lung, cholangiosarcoma of the liver, and soft tissue sarcomas at 1 and 5 ppb levels, and carcinomas of the ear duct, kidney and sk[illegible] as well as soft tissue sarcomas and testicular tumors at the 5 ppt [illegible]. In more large scale carcinogenicity tests by DOW Chemical, [illegible] are generally confirmatory of the Van Miller studies, TC[illegible]inistered orally to rats at levels of 0.001, 0.01, and 0.1 [illegible] et al., 1978). Carcinomas of the liver, lung, palate and [illegible] were induced at the highest dose levels, neoplastic liver nodules at the intermediate dose level, and toxic liver effects at the lowest dose level tested.

These results have been confirmed and extended in recent NCI carcinogenicity tests in mice and rats, conducted by the Illinois Institute of Technology, at concentrations of 0.01, 0.05, or 0.5 ug/kg/week for rats and male mice, and 0.04, 0.2, or 2.0 ug/kg/week for female mice, respectively (NCI, 1980). Thyroid adenomas and carcinomas were induced in the higher dose groups of the rats, and also liver cancers in the highest female dose group. Similar tumor patterns were induced in the mice.

2. In Humans

The toxic effects of dioxin alone or in conjunction with TCP, 2,4,5-T and related chlorophenoxy compounds, have been investigated or recognized under a wide range of conditions and circumstances. In addition to exposures in South Vietnam, these include about 8 occupational accidents, some involving both workers and the surrounding community; about 15 occupational exposures from handling contaminated intermediates, such as TCP, within plants; occupational exposures during agricultural, forestry or rights-of-way application; community exposures from dioxin contaminated waste oils, such as in Times Beach, Missouri; and presumed community exposures in Midland, County, Michigan in proximity to the DOW Chemical facility whose soil is heavily contaminated with dioxin. The symptomatology and effects induced by dioxin reflect a wide range of factors including dosage or exposure levels, duration of exposure, presence of other toxic chemicals and contaminants, and age, sex, and reproductive status of exposed populations.

a. chloracne

There appears to be considerable misunderstanding as to the role of chloracne in Agent Orange disease, with particular regard to its alledged obligatory or pathognomonic value as an index of exposure (Epstein, 1980). As pointed out in this testimony, there is marked species-specificity in experimental animals for the acneigenic effects of dioxin; mice and rats being resistant and rabbits and monkeys being sensitive. Chloracne has protean manifestations ranging from chronic pustular cystic, follicular, acneiform, hyperkeratosis, and blepharo-conjunctivitis, to milder reversible forms with minimal residual scarring, pitting, hyperpigmentation, depigmentation, and hypertrichosis. The chloracne is generally, but not invariably, associated with a wide range of other symptoms and objective signs of multi-system disease.

Chloracne has been recognized since the 1890s as an occupational dermatosis in workers handling chlorinated phenols, diphenyls, and napthalenes. These lesions attracted the attention of dermatologists, whose reports indicted an interest restricted to the skin. There is thus a probability that a bias has developed, particularly in the early literature, in the over-emphasis of the dermatological aspects of dioxin and in the failure to recognise systemic toxicity in the absence of overt skin disease. This may well have resulted in substantial under-reporting of dioxin toxicity. These considerations have been further strengthened by reports of dioxin-induced systemic disease in the absence of chloracne (Bleiberg et al., 1964; Oliver, 1975; Scarisbrick & Martin, 1981; Martin, 1983) and by the absence of a history of chloracne in cases of soft-tissue sarcoma in workers exposed to phenoxy herbicides and chlorinated phenols (Honchar & Halperin, 1981; Johnson et al., 1981; Moses and Selikoff, 1981). Furthermore, of a group of children and young adults with acute burnlike skin lesions following the Seveso accident, only 12 subsequently developed chloracne, which was mild and reversible (Reggiano, 1980). Clearly, chloracne is not an obligate determinant of dioxin toxicity.

Of interest in this connection is the dismissal by the VA of most skin condition claims for compensation, irrespective of their signs and symptoms and exposure history, on the grounds of the absence of classic chloracne. Insistence by the VA on specific proof of exposure and on evidence of classic chloracne, irrespective of other symptoms and evidence of disease, is thus inconsistent with the scientific realities and inappropriately shifts the burden of proof, and a virtually unattainable burden at that, to the individual veteran. This extreme position, whether reflecting lack of familiarity with scientific literature, bureaucratic inertia, or fear of opening the floodgates of compensation claims, is in interesting contrast with the awarding of compensation for service-related disability to veterans contracting multiple sclerosis within seven years of the termination of service, even though the etiology of this disease is poorly understood and its possible relation to military service is unknown.

b. Multi-System Disease

Dioxin, as well recognized in the clinical and toxicological literature, is a potent multi-system toxic agent producing a panoply of acute and delayed effects, many of which can progress to the chronic. Recognized clinical symptoms include asthenia, muscular weakness, pains in limbs and joints, insomnia, photosensitivity, nausea, vomiting and diarrhea. Recognized signs include abnormalities in liver function, porphyria cutanea tarda, peripheral neuropathy, elevated blood triglycerides and cholesterol, and psychological and personality changes; additionally, an excess of cardiovascular deaths from coronary disease in a Dutch TCP accident clean-up crew, and also a case of rapidly progressive atherosclerosis in a TCP accident clean-up crew, and also a case of rapidly progressive atherosclerosis in a TCP worker have been reported. This disease complex is generally consistent with that recognized in preliminary surveys of Vietnam veterans (Bogen, 1980; Stellman & Stellman, 1980), and with those recognized following occupational accidents and exposures. Other major symptom categories include gastro-intestinal, neurological, and psychological. A history of alcohol intolerance, is not uncommon and appears suggestive of liver dysfunction.

c. Cancer

With the exception of recent Swedish studies, the epidemiological literature on cancer rates in occupationally exposed workers or in workers involved in TCP or related accidents is too inadaquate, particularly in size of cohorts and duration of follow-up, to sustain valid negative inferences. An apparent example is a follow-up study on 121 workers developing chloracne, and systemic toxicity, following the 1949 Monsanto accident in Nitro, W. Va. (Zack and Suskind, 1980). The report appears to exclude exposed workers who did not develop chloracne, and fails to provide basic information on age distribution and employment history. Furthermore, the controls were based on the general population rather than on similar industrial populations with the resulting likelihood of underestimating mortality rates from cancer and other diseases. While the study recognized the small cohort size and the number of observed deaths, it was concluded that there was no apparent excess of cancer mortality. Contrary to these conclusions, however, the cited data clearly demonstrates excess mortality from lymphoma and leukemia.

Additionally, and of critical interest, are the findings among these workers that of two deaths from soft tissue sarcomas, only one was reported to have chloracne.

There are also some interesting cohort studies which provide suggestive evidence. Illustrative, is the follow-up of the 1953 Ludwigshafen accident in a TCP plant, in which a 7-fold excess in stomach cancer over expected rates was noted (Thiess & Frentzel-Beyme, 1977). A statistically significant excess of stomach cancer has also been reported in an updated analysis of an initially questionable study in a cohort of Swedish railroad workers exposed to phenoxyacetic acids and amitrole over 10 years previously (Axelson et al., 1979); the excess was found in the total cohort and in the phenoxy subcohort. In a 5 year follow-up of 55 subjects in a cohort of 78 workers involved in an explosion in Spolana, Czechoslovakia in 1965, 2 cases of lung cancer were noted versus 0.12 expected (Jirasek et al., 1973).

In a recent case control study in a population in Northern Sweden with a high proportion of paper pulp, forestry, and sawmill workers, a relative risk for soft-tissue sarcomas in workers exposed primarily to 2,4,5-T and 2,4-D was more than 5 times greater than in unexposed individuals; this finding was statistically highly significant (Hardell & Sandstrom, 1979). Even higher risks were found in groups exposed to other chlorophenols contaminated with chlorinated dibenzofurans. This study was confirmed by similar findings of statistically significant excess risks for soft tissue sarcomas in other populations exposed to chlorophenols in a different area in Southern Sweden (Ericksson et al., 1979). These studies have also demonstrated excesses of other cancers including malignant lymphomas.

These findings are consistent with reports on a 40-fold excess of soft tissue sarcomas in dioxin exposed workers including those from DOW and Monsanto (Honchar & Halperin, 1981); with an approximate 800% increase in soft tissue sarcomas in female residents in Midland County, Michigan, from 1970-78, (compared to 1950-59), and with reports on 3 cases of soft tissue sarcomas in 3 Vietnam veterans heavily exposed to Agent Orange, none of whom had any prior history of chloracne (Sarma & Jacobs, 1981). The association of dioxin exposure and soft tissue sarcoma is of critical significance. Soft tissue sarcomas are exceptionally rare and highly lethal malignancies whose background incidence in males aged 25-40 is only 13 per million.

In addition to the reports on soft tissue sarcomas in Vietnam veterans, there are also growing numbers of case reports possibly suggestive of an apparent excess incidence of cancer in various groups of Vietnam veterans, although these data have not yet been subject to epidemiological analysis. The incidence of testicular cancer appears high, even allowing for selection bias. In one group of about 5000 plantiffs, approximately 200 testicular cancers, mainly seminomas, have been recognized. In the absence of data on age distribution and date of first diagnosis (as this tumor is associated with prolonged survival), it is difficult to estimate whether this incidence is excessive. Seminomas are rare, and fewer than 2,000 are diagnosed annually in the entire white U.S. population; the incidence in blacks is considerably less. There are also suggestions of an increased incidence of lymphomas and leukemia.

d. Reproductive Toxicity

There are a growing number of case reports on reproductive toxicity, including impaired spermatogenesis, infertility, and birth defects, among Vietnam veterans. The defects reportedly tend to be bizarre, mainly involving extra or missing limbs or limb deformities, and are also sometimes associated with mental retardation. Experimentally, dioxin induces a wide range of adverse reproductive abnormalities, including teratogenicity, and gonadotoxic effects in male rodents, chickens, and monkeys. However, the scientific basis of paternally-mediated teratogenicity is still obscure. A large scale NIEHS study on male reproductive effects in mice, including studies on dominant lethality was essentially negative. A negative standard dominant lethality test does not, however, eliminate a genetic basis for paternally-mediated birth defects. There is significant literature on the genetic transmission of sperm abnormalities induced by chemical mutagens. This has been substantially strengthened by the recent demonstration of neurobehavioral deficits in the $F_1$ progeny of cytoxan-treated male rats (Adams et al., 1981). Moreover, the unresolved problem with birth defects in veteran's children is the reality of the apparent excess, not current inability to comprehend its basis. Of interest in relation to paternally/mediated effects are the recent findings of an increased failure in conception rates in the wives of dioxin exposed workers in the middle 1970s (Townsend et al., 1982).

The study on wives of dioxin exposed workers also yielded suggestive evidence of excess birth defects which were also found in wives of DOW workers without recorded occupational exposures to dioxin but residing in Midland County, Michigan, from 1971-74, suggesting the importance of community exposure to dioxin. These data are also consistent with excesses noted in comparisons of birth defect rates based on birth certificates of residents in Midland County, Michigan, in contrast with residents of the state of Michigan over the same four year period. Of greater significance however, are the findings of major excesses of cleft lip and cleft palate in Midland County births from 1971-74 (Houk, 1983; Poole, 1983).

D. Comments on H.R.1961

H.R.1961 establishes a presumption of causality between Agent Orange exposure and three categories of adverse health effects; soft tissue sarcomas, porphyria cutanea tarda, and chloracne, and for other unspecified disease conditions as determined by the disretion of the VA Administrator. This restriction to the specified disease categories is, however, at variance with an extensive body of toxicological, clinical and epidemiological data on the effects of dioxin, and as such improperly excludes a wide-range of dioxin induced diseases. The discretion afforded the Administrator to recognize and compensate other dioxin induced diseases appears to reflect an unduly optimistic faith in the agency's ability and interest to take such steps.

An alternative concept which this subcommittee may wish to consider is the establishment of a non-rebuttable presumption of causality for the specified diseases and for those other diseases which are consistent with the scientific literature on dioxin toxicity. Such presumptions would be still further strengthened by the absence of other defined and exclusive causalities for the disease in question.

For Agent Orange disease not only is a high of absolute level of proof inappropriately demanded by the VA, but also the burden of proof is shifted to the veteran. While H.R. 1961 is a commendable step in the direction of reducing this heavy burden, it fails to reflect the major inconsistencies with established practices in compensating other diseases such as multiple sclerosis (MS), if manifested within 7 years of discharge, even though there is virtually no scientific data relating MS to service, in contrast with the substantial data relating the Agent Orange disease complex to dioxin exposure. Such inconsistencies raise the suspicion that constraints to the recognition of the extent of Agent Orange disease and need for its equitable compensation may reflect the political and economic considerations, rather than scientific considerations. Such constraints are often manifest in recommendations to postpone further consideration of compensation pending results of further studies; apart from the striking contrast of a wide range of Federal acts of restitution in instances when data on causality are much less well developed than for Agent Orange disease, such as compensation for MS and the Times Beach buy-out. Many of the studies in question are unlikely to yield other than invalid or equivocal data, for reasons of design defect, or for such problems as attempted retrospective quantitative exposure, which in turn would serve as still further alledged justification for yet more postponement and study.

Illustrative of such problems is the Ranchhand study which is unlikely at this stage to yield adaquate information on mortality and chronic disease with particular reference to cancer. Additionally, there are major problems in attempting to extrapolate exposure patterns and other data from Ranchhand personnel to the ground troops, who operated under conditions involving confounding variables, such as heat, stress, fatigue, and were often unable to shower or change their contaminated clothing for long periods.

Such considerations were clearly articulated by Joan Bernstein, Chair of the original Interagency Work Group on Phenoxy Herbicides and Contaminants and General Counsel of H.H.S., who testified in September, 1980 before the House Committee on Veterans Affairs that the anticipated research findings "will not tell us what elevation of risk a veteran's illness should be deemed service connected...(and) will not and cannot by itself answer questions that seem to us to be fundamentally ones of broad social policy..."

This subcommittee may also wish to consider the inconsistencies between heavy burdens of proof still imposed on the Vietnam veteran with recent unprecedented governmental action in buying out the Times Beach community on the basis of grave imminent hazard posed by exposure to dioxin. Finally, Congress should consider the belated need to shift the burden of compensation for Agent Orange disease from the U.S. taxpayer to DOW Chemical, who while publicly asserting in 1964 that 2,4,5-T was "absolutely non-toxic to humans or animals," deliberately suppressed substantial information on the presence and toxicity of its dioxin contaminant.

## E. Conclusions

1. Concerns on dioxin-induced disease among U.S. veterans have recently burgeoned. They now involve all major veterans' organizations, besides many new ones which have been created to deal with these problems, the Congress, the White House, a wide range of Federal agencies, State governments, the courts and the general public.

2. There is now growing and substantial evidence of a clear consistency between the wide range of toxic effects induced by dioxin in experimental animals of various species and those observed in a wide range of exposed human populations, including [redacted] occupational and Vietnam veterans. This consistency relates to multisystem disease, reproductive toxicity, and carcinogenicity. Overemphasis on the significance of chloracne as an allegedly obligate determinant of dioxin exposure has limited previous recognition of a wider range of other toxic effects. Recent reports of the very rare and highly lethal soft-tissue sarcomas in three Vietnam veterans have also provided a critical link with a wide range of studies demonstrating major excesses of soft-tissue sarcomas in groups occupationally exposed to dioxin. Such data, together with qualitative evidence of exposure have created the strong presumption of causality in Agent Orange disease.

3. Current objections to evidence of causality in Agent Orange disease have been expressed by insistence on quantitating past exposure and on continued prospective studies. Reacting to such objections, and responsive to national concerns, are recent trends in the Congress to reduce the burden of proof on the individual veteran to establish the scientific validity of claims for Agent Orange induced disease, and to shift this burden in the direction of the government. While unduly restrictive, H.R.1961 is clearly a step in the right direction of reflecting such concerns, for which the sponsors of the bill should be commended.

4. Besides extending the scope of H.R.1961, this subcommittee may wish to examine the evidence relating to the justification and

feasibility of shifting the burden of restitution from the government to the manufacturers of Agent Orange in view of their suppression of information on the presence of the highly toxic dioxin contaminant in Agent Orange.

Thank you very much, this concludes my testimony.

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PREPARED STATEMENT OF TESTIMONY BEFORE THE SUBCOMMITTEE ON COMPENSATION, PENSION AND INSURANCE LEGISLATIVE HEARING ON H.R. 1961, VIETNAM VETERANS AGENT ORANGE RELIEF ACT, ON APRIL 26 AND 27, 1983 - ROOM 334, CANNON HOUSE OFFICE BUILDING, WASHINGTON, D.C.

April 22, 1983

My name if Jerry Lee Simmons, M.D. I was born 29 October, 1939 in Maumee, Ohio. My Social Security number is 296-34-2519.

I graduated from Bowling Green State University at Bowling Green, Ohio in June, 1962, and attended medical school at The Ohio State University College of Medicine, Columbus, Ohio, graduating in June, 1967. I was ordered to active duty from the U.S. Army Reserves on the 27th of June, 1967, and served a rotating internship at Brook General Hospital in Ft. Sam Houston, Texas from July 1967 to June 1968. During the Tet Offensive in 1969, I was an intern on the orthopedic service where we took care of over 150 bed patients and 750 patients on the service at any one time. Many of these patients were within 24 hours of their war wounds suffered in the VietNam conflict.

Following my internship, I trained at the U.S. Medical Field Service School (AMEDS) Officer Basic Training in 1968 followed by U.S.A. Aviation School at Ft. Rucker, Alabama in 1968. In October of 1968 I was ordered to VietNam, where I served until October 1969. For the first few months I was assigned as a flight surgeon to the 159th Heavy Helicopter Battalion stationed in Phu Bai in Northern ICORE. The 159th Heavy Helicopter Battalion is part of the 101st Aviation Group of the 101st Airborne, now Air Mobile Division. Midway in my tour of duty, I was promoted to the Group Flight Surgeon and moved to Camp Eagle with 101st Aviation Group of 101st Airborne Division.

My duties in VietNam included care of aviation and non-aviation personnel assigned to the 159th Heavy Helicopter Battalion and later all personnel assigned to the 101st Aviation Group Headquarters and assigned Pathfinder Unit. My duties included going on perimeter patrols as medical support, investigating aviation accidents and losses wherever they occured throughout ICORE and Laos, and going into hot landing zones with the Pathfinders to assess and support their medical needs. During this assignment we spent considerable hours in the field, both in the air and on the ground, including ground excursions into many defoliated areas in the Ashau Valley, Laos and throughout Northern ICORE. During that time there was considerable perimeter spraying with defoliants. Perimeter patrols, of necessity, were exposed to these defoliants on a regular basis.

The units were frequently sprayed with various insecticides.

As officer in charge of the health needs of our battalion, and later the entire aviation group, I insisted that each member faithfully take their Dapsone anti-malarial tablets, as well as quinidine. The quinidine was taken once a week, but Dapsone was taken every day, including a 60-day period after return state-side.

The drinking water was heavily chlorinated. This is another potential source of toxic compounds which, to my knowledge, has not been fully evaluated.

Many of us were exposed to various tropical diseases, including various forms of hepatitis. There was also moderate to wide spread alcohol and drug ingestion. The effect of the combination of alcohol and/or other drug ingestion, including medicinal drugs such as Dapsone, in combination with various pesticides, chlorinated compounds and herbicides is unknown.

It is my understanding that Dapsone, at the time it was given in VietNam, was an experimental drug. We, the medical officers in charge of the health of the soldiers, were not informed that this was an experimental drug. In my opinion, from my experience in VietNam, Dapsone undoubtedly saved many lives. In several instances, 101st Airborne soldiers taking Dapsone would not get malaria while 1st Marine Division marines in the next valley would have a high incidence of malaria. Nevertheless, the effects of the "little white pill" - Dapsone, in combination with other substances we were exposed to in VietNam should be further investigated.

During my tour of duty in VietNam we participated in many MEDCAP programs. These medical civic action programs were designed to promote good will by American solders, including physicians, and coreman working with and teaching South VietNamese civilians to better care for their own health problems. We observed many strange and exotic diseases, including cholera, rampant tuberculosis, many kinds of parasitic disease, and two separate plague epidemics. Many of the children and adults had various skin problems which, in retrospect, may have been chloracne.

We also noted skin problems in many of our American soldiers, especially those who spent considerable time in the field and/or on perimeter duties. These skin problems, in retrospect, could have been chloracne.

After serving my tour of duty in VietNam, I was assigned to Ft. Knox, Kentucky as the Post Flight Surgeon. We served at this post until the 26th of June, 1970, when I was honorably discharged from the United States Army Reserves.

I was fortunate enough to obtain a residency position in the Department of Pathology at the University of Michigan, where I trained from 1970 to 1974. Part of this training included tours of duty at the Veterans Administration hospital. From June of 1974 until May 1981, I was employed by the Veterans Administration Medical Center in Ann Arbor, Michigan. During that time period I became board certifed in anatomic and clinical pathology (1974) and became a fellow of the American Society of Clinical Pathologists and the College of American Pathologists. I also belong to the American Medical Association, State Medical Association, and District Medical Association, the American Association for Clinical Chemistry, on the subcommittee on Animal Clinical Chemistry, and I am an honorary member of the Michican Society of Histotechnologists. While at Michigan, I developed several areas of interest, including comparative pathology, gastrointestinal pathology, and muscle and peripheral nerve pathology. In 1975 and 1976, I was the interim director of the neuro-muscular pathology laboratory at Michigan State University and served as an Assistant Professor at Michigan State University, as well as instructor and later Assistant Professor at the University of Michigan.

In 1975 I became involved in the scientific study of P.B.B., a fire-retardant which had contaminated the cattle feed in Michigan. This substance quickly spread to most Michigan residents via milk and milk products. I collaborated with Dr. Tom Corbett, an anesthesiologist in the study of the effects of P.B.B. on mice and gerbils. This collaboration resulted in several scientific presentations and a publication.

During this same time frame, we became increasingly aware, perhaps due to our interest in environmental contaminants, of VietNam veterans who had multiple complaints which they felt were due to Agent Orange. I became superficially involved with several veterans organizations in Michigan, including Citizen Soldier. Through this relationship I became aware of many health problems VietNam veterans were suffering. These health problems included birth deformities, multiple gastro-intestinal complaints, emotional disturbances, skin diseases and circulation disorders. Unfortunately many of these cases were hearsay or one of a kind occurrences. There seemed to be an increase in carcinomas in a young age group. In 1978 and 1979 I suggested we save fats from autopsies from VietNam patients for possible dioxin. We attempted to find commercial sources for this evaluation. We found the cost was prohibitive, at least $1,000.00 per sample. We were also discouraged from saving fats because there was no mechanism for proper storage or a central repository (such as A.F.I.P.). As you are aware, at the present time, the Armed Forces Institute of Pathology requests all surgical specimens from VietNam veterans be sent to them as a registry. However, to my knowledge, at the present time there is no concentrated effort to save and/or evaluate fats for herbicides from VietNam veterans undergoing surgery or autopsy.

In 1981 we decided to move to Sioux Falls, South Dakota in the position of Clinical Chief of the Laboratory Service. This move was made because a new Chairman of Pathology, whose interests were divergent from mine, had been appointed at the University of Michigan. If felt there was greater opportunity for advancement by moving to South Dakota. At present, I work for the Laboratory of Clinical Medicine, which has a contract with the Veterans Administration Medical Center in Sioux Falls to provide the pathology services. Because of my previous Veterans Administration experience, and my background as a probable Agent Orange exposed veteran, I have been assigned to cover the position of Clinical Chief of Laboratory Service.

In general, I feel the Royal C. Johnson Veterans Administration Medical Center in Sioux Falls, South Dakota is an excellent facility. The Director, Chief of Staff, Chiefs of Bed Services, administrators and employees have an excellent attitude toward VietNam veterans and are willing to work with them in any way possible to help solve their problems. The VietNam veterans I have talked to who have been examined at our facility felt their examination was thorough and they were handled with courteousness and professionalism. As of 8-9-82, we have a cumulative total of 868 Agent Orange appointments, of which 82 had dermatology references, 419 were South Dakota veterans, 371 were Minnesota veterans, and 64 were Iowa veterans. There were 14 others. Ten (10) of the Agent Orange veterans had an examination with C&P, and 17 patients had examinations as in-patients.

We have two excellent veterans out-reach centers; one in Sioux Falls and another in Sioux City. I have talked with personnel from both of those centers about Agent Orange and its scientific and medical aspects.

We provide psychiatric, psychological, and social service counselling for VietNam veterans, as well as veterans in general. There is a substance abuse program in our hospital which is unfunded, but which, nevertheless, does an excellent job in providing services to both VietNam and non-VietNam veterans. Those patients needing peer counselling are referred to the veterans out-reach centers.

During the past year I have had the opportunity to attend the Second International Agent Orange Conference held in October in Arlington, Virginia. This conference was rather enlightening, as a cross-section of "experts" presented various scientific and social topics concerning Agent Orange. In conjunction with this conference, a two volume set on the review of literature on herbicides, including phenoxy herbicides and associated dioxins, volume I and II was prepared by the Veterans Administration under the leadership of Barclay M. Shepard, M.D. I have found these publications to be most useful in answering most of the scientific inquiries concerning Agent Orange. During the conference, it became increasingly apparent that even the experts did not know the effects of Agent Orange or any other herbicides, pesticides or other substances used in VietNam. This area certainly deserves much further study. One of the questions I asked the panel was why we had been discouraged to save fat specimens from VietNam veterans undergoing surgery or autopsy. A satisfactory answer was not forthcoming other than no one would know what the results meant anyway. It was brought out that the samples analyzed from the Ranch Hand evaluation showed some of the controls who had never been to VietNam had higher levels of dioxins than those who were presumably heavily exposed.

In discussions following, we pointed out that it would make a lot of difference in total exposure if personnel was to able to quickly shower following contamination. This would be possible for Air Force personnel but impossible with Army personnel on patrols in the "boonies."

Another fact that became apparent during the conference was dioxin is a very dangerous substance. Researchers using dioxins describe their biologic safety set-ups. This type of facility is well beyond the reach of most investigational institutions, certainly including ours in Sioux Falls.

During the past several years there has been periodic publicity regarding dioxin, a contaminant of Agent Orange. Recently the Times Beach episode, and the alleged dioxin contamination of water sources in the Midland, Michigan area, has resulted in considerable coverage by the media. The medical literature contains very little hard data or conclusions regarding the results of human exposure to dioxins. A two volume review of literature prepared by the Veterans Administration, Department of Medicine and Surgery, was completed in 1981. Another viewpoint is expressed by Archie B. Blackburn, M.D. in his article, "Review of the Effects of Agent Orange: A Psychiatric Prospective on the Controversy," published in "Military Medicine," Volume 148, April 1983.

Most Americans seem unaware that during the time period 1961 through 1969, 44 million pounds of 2, 4, 5-T were actually sprayed in VietNam while 78.1 million pounds were used in United States for weed control, railroad rights-of-ways and forest-related vegetation control. It should be pointed out that military spraying utilized 27 times more herbicide per unit area than most of the domestic operations (Harrington, J. F.: Herbicide Orange Surplus. Science, 180:1320 (1973). Those of us who have contact with VietNam veterans, counselors, and health professionals caring for VietNam veterans can relate many cases of young veterans with unusual cancers, birth defects in their children, multiple neurologic and psychiatric complaints and skin rashes. Many veterans complain of vague gastrointestinal disorders. Some develop disorders of the immune system manifest by either hypersensitivity phenomenon or vascular disturbances, such as stroke or thrombosis of extremities. Unfortunately, these cases are rarely reported. The association of these diseases with Agent Orange exposure is difficult, if not impossible, to prove.

Many states, veterans groups, and governmental agencies including the Veterans Administration have attempted registries of Agent Orange exposed veterans. The Armed Forces Institute of Pathology has requested tissues on patients with VietNam experience. Evaluation of these cases has, so far, not shown any clustering of diagnoses (Dr. Nelson Irey, personal communication).

The 1976 Seveso incident has been well documented. The symptoms of exposed people are very similar to those expressed by VietNam veterans including loss of hair, liver and kidney disease, hypothyroidism, neurologic disorders including loss of hearing, smell and taste, depression, lack of vigor, sleep disorders, emotional instability and decreased libido. Many people develop skin rashes and had impaired immune reactions. Follow-up studies so far have not revealed increase in neoplasms, however, it is too early to make valid scientific conclusions.

Most studies on humans, primarily from questionnaires, indicate increased neoplasms including kidney, testicular, lymphoma, and soft tissue sarcomas. Unfortunately these studies are not well controlled and no valid scientific conclusions have resulted.

Animal studies show various responses, depending on the species studied. These include skin hyperplasias (chloracne), immune suppression and involution of lymphoid organs, birth defects, liver lesions including neoplastic proliferation and porphyria. The effects seem, in part, dose related.

In my opinion, the physical effects of Agent Orange other than chloracne are still unproven. In all fairness, at this point it cannot be stated with certainty that these symptoms are or are not caused by Agent Orange or other chemical exposure. It should be pointed out there were many other factors in VietNam veteran exposure, including pesticides, herbicides, other medications such as Dapsone, infectious diseases including malaria, hepatitis, and melioidosis, highly chlorinated water and local drugs of unknown composition.

From my prospective as a physician, I feel the health problems VietNam veterans are experiencing are real. At the present time I do not feel anyone can say whether or not these health problems are secondary to Agent Orange or other chemical exposure which may have occured in VietNam or, for that matter, here in the States. Whatever the cause, these health problems must be recognized and treated. The short and long range effects of the VietNam experience must continue to be studied in all of its ramifications. This includes incidence of birth defects, cancer development in VietNam veterans, cardiovascular complications, and stress-related conditions.

At the present time, most VietNam veterans, including myself, feel that only combat-exposed peers really understand our problems and feelings. In this regard the Vet Centers throughout the United States should be fully funded. Increasing efforts must be made to identify, counsel, and take care of the needs of our VietNam veterans, both now and in the future.

JERRY L. SIMMONS, M.D.
Clinical Chief, Laboratory Service

# STATEMENT OF
DOROTHY L. STARBUCK
CHIEF BENEFITS DIRECTOR
BEFORE THE
SUBCOMMITTEE ON COMPENSATION, PENSION AND INSURANCE
COMMITTEE ON VETERANS' AFFAIRS
HOUSE OF REPRESENTATIVES
APRIL 27, 1983

Mr. Chairman and Members of the Subcommittee:

I am pleased to be here this morning to present the views of the Veterans Administration on H.R. 1961, 98th Congress, the proposed "Vietnam Veterans Agent Orange Relief Act." Mr. Chairman, I share with you and other members of Congress the desire for a meaningful Federal response to the fears of veterans who served in Vietnam that their exposure to Agent Orange may have long-term adverse effects on their health. However, we consider the approach taken in H.R. 1961 inadvisable given the present state of scientific knowledge.

The controversies arising from the Government's use of Agent Orange in Vietnam are not yet resolved. Before turning to the Veterans Administration's observations

Veterans Administration's observations concerning the several issues raised by H.R. 1961, I would like to emphasize that the potential cost of paying compensation based on any Agent Orange-caused disabilities played no part in our deliberations on this measure. The Federal Government, since its beginning, has fulfilled its sacred obligation to veterans disabled in the line of duty and will continue to do so.

Mr. Chairman, the devastating wars of this century, and the need to maintain peacetime forces in order to assure the defense of our Nation, have been accompanied by legislative and programmatic developments intended to assure that no veteran's reasonable claim to compensation is denied. This is true whether the disability results from a combat wound, service-incurred disease, in-service accident, psychological trauma resulting from combat or other conditions of military service, or exposure to a substance known or later discovered to have adverse health effects.

We are immensely proud of our Agency's record of achievement. It can safely be maintained that our compensation program is the finest in the world, both in terms of the number of veterans we serve and in the amount of benefits paid. Moreover, the American people--who fund this program with their taxes--have given it overwhelming support, as has the Congress of the United States.

The preservation and integrity of the compensation program are among the highest priorities of the Veterans Administration.

There are certainly many veterans suffering from illnesses they ascribe to exposure to that herbicide, especially its contaminant dioxin. Although scientific evidence is lacking, there are persons in the medical and scientific communities who contend that exposure may lead to a host of disorders that appear long after the exposure has ceased. There are also organizations and individuals who believe very sincerely that the Veterans Administration has not responded adequately to the issues involved.

As guardians of the public trust, Congress and the Administration share, I believe, a commonality of aims respecting these issues. The compensation program must be attuned to justifiable conclusions about the connection between Agent Orange exposure and disorders possibly arising from that exposure. At the same time we must do our best to avoid taking steps that have the potential for undermining the program's credibility and legitimacy because of inconclusive scientific evidence. I know that you and other Members will give careful and thorough consideration to the bill, keeping in mind the commonality of aims to which I have previously alluded.

H.R. 1961 is intended to assist veterans who served in Southeast Asia during the Vietnam era establish entitlement to service-connected disability compensation if they are currently suffering from one of the disorders specified in the bill. It would do this by amending section 312 of title 38, United States Code, in order to provide for a special presumption of service connection applicable only to these veterans.

Mr. Chairman, the bill is based on the premise that each of the specified disorders, no matter how long after military service symptoms appear, can be attributed to exposure to a phenoxy herbicide in service. During the period 1962 to 1971, phenoxy herbicides, including Agent

Orange, were used in Vietnam. As I have noted, H.R. 1961 is an effort to respond to the widespread concern that exposure to Agent Orange, especially its contaminant dioxin, may have long-term adverse effects on veterans' health.

Authority to award compensation on the basis of the presumption provided for in the bill would terminate one year after submission to Congress of the comprehensive epidemiological study mandated by Pub. L. No. 96-151. This "sunset" provision is analogous to the sunset provision applicable to VA health care for certain disorders possibly associated with phenoxy herbicide exposure, authorized by Pub. L. No. 97-72. Both sunset provisions recognize the current uncertainties as to the long-term effects of exposure.

The Agent Orange controversy, as it relates to individual veterans' compensation claims, involves two basic questions: (1) whether the veteran was exposed, and (2) whether the veteran's disability results from the exposure. H.R. 1961, it should be noted, does not require any evidence of exposure; it would afford the presumption to any veteran who served in Southeast Asia during the Vietnam era (1964-1975). We have previously made public our decision to resolve the issue of exposure in a manner favorable to veterans; unless there is affirmative evidence to the contrary, we are prepared to presume exposure if a veteran served in Vietnam during the relevant period. This policy, prompted by the lack of a definitive method for identifying exposed individuals, is consistent with our longstanding policy of giving veterans the benefit of the doubt.

There may be, however, some cases in which affirmative evidence refutes even the possibility of exposure, and, therefore, our policy is necessarily qualified. The lack of any similar qualification in H.R. 1961, in our view, is unjustifiable. We observe also that affording the presumption to veterans who served in Southeast Asia--a far broader region than Vietnam, embracing areas where no phenoxy herbicides were used--inappropriately expands the category of veterans intended to be benefited.

Our principal concerns, however, relate to the concept of an open-ended presumption that would be established by the bill and to the conclusions it embodies as to the specific disorders chloracne, porphyria cutanea tarda (PCT), and the several malignancies grouped as soft-tissue sarcoma. (The bill would also authorize presumptive service connection for additional disorders, provided for by regulation, that "medical research" has shown "may be" attributable to chemical exposure or environmental hazards or conditions. This requirement is vague--rules creating such presumptions should be based only on well-accepted, scientifically valid findings--and also unnecessary in view of the Administrator's current authority to issue regulations.)

The post-service presumption periods provided for in section 312(a) of title 38 are appropriate for chronic diseases whose inception in service may not be recorded because the development of pathology is gradual and insidious. They are justifiable, Mr. Chairman, when reasonably supported by medical knowledge as to the pathological courses of the particular diseases.

Congress has wisely set time limits on these presumptive provisions; unless symptoms of the disease appear within a specified period of time after service, the presumption is not available. The section 312(a) presumption, together with the time limits, assures that no veteran's reasonable claim is overlooked but also does not dictate grants of service connection when there is no evidence of service incurrence and it is not reasonable to infer service origin.

Reputable studies have concluded that dioxin exposure may result, within a relatively short period, in chloracne. PCT resulting from exposure also appears within a few weeks. On the other hand, no studies have shown that exposure results in the initial appearance of these disorders after lengthy delays. Mr. Chairman, our current authorities are adequate, without the need of a presumption, to award service connection and compensation, if appropriate, in cases of chloracne or PCT appearing within expected time limits after the exposure. Requiring us to award service connection for initial occurrence of these disorders long after the exposure incidents is, we believe, unjustifiable in the absence of any evidence indicating they are latent effects of exposure.

As I have noted, individuals in whom these relatively rare disorders appear begin to suffer symptoms soon after exposure, ordinarily within days or weeks. Chloracne is a skin disorder caused by exposure to certain chlorine-containing chemicals, including dioxin. In its more serious manifestations, it causes discomfort and disfigurement. Most cases clear up within a year or two after the exposure ceases, but in a few, the disorder persists. The Veterans Administration acknowledges that chloracne can result from exposure to Agent Orange during service in Vietnam and has established procedures to assure careful and liberal consideration of all claims based on this disorder.

Since 1978, we have awarded service connection in 1,225 skin-disorder cases involving veterans who served in Vietnam. We have scrutinized more than 3,000 claims for service-connected benefits to determine whether there are indications of chloracne. Those cases in which it was believed this diagnosis was at least possible were further reviewed by a VA dermatologist, and 13 have been examined in person by dermatologists at prestigious private clinics.

Although all of these cases involve skin disorders of various types and all involve veterans who served in Vietnam, only one case of possible chloracne has been identified. We will, of course, continue our investigations of this issue.

H.R. 1961 would also extend presumption of service connection for "chloracneform lesions." This is a term not found in medical or scientific literature, but can be taken to mean "lesions resembling chloracne." As certain common skin disorders may resemble chloracne, this term is overly broad and would, we believe, cause unnecessary confusion.

PCT, an uncommon liver disorder, can be triggered by exposure to various chemicals including alcohol. There is no evidence that PCT is a latent effect of exposure. Each attack ordinarily subsides in about a year after contact with the chemical ceases, but prolonged exposure, as in chronic alcoholism, may cause permanent damage to the liver. An attack of PCT induced by Agent Orange or exposure to any other chemical during service in Vietnam years ago would not be expected to impair a veteran's health today. As is the case with chloracne, we regard our current authorities as fully adequate to assure proper consideration of PCT claims based on exposures during military service. As a technical matter, the proper application of section 313 of title 38, United States Code, making section 312 presumptions rebuttable if there is evidence of an intercurrent cause, would reduce the likelihood of awards of service connection based on the PCT presumption, if enacted.

Mr. Chairman, the issue as to whether the malignancies grouped as "soft-tissue sarcomas" result from phenoxy herbicide exposure presents a problem of far greater complexity. There is considerable uncertainty in the scientific community on this issue. Advocates of the belief that exposure "causes" soft-tissue sarcoma generally cite studies involving cancer victims believed to have been exposed to phenoxy herbicides whose first symptoms appeared long after the exposure. Because it is well established that exposure to radiation and other agents like asbestos and benzene may result in the latent development of malignancy, these advocates reason by analogy that phenoxy herbicide exposure "causes" soft-tissue sarcoma. The vital question is, therefore, the weight that should be given to the studies they cite.

"Soft tissue sarcomas" are a group of malignant tumors, or cancers. Any sarcoma arises in a body cell that does not cover a body surface, form glandular tissue, or line certain body cavities. "Soft tissue" excludes sarcomas in "hard tissues" such as bone or cartilage. Hence, soft tissue sarcomas arise from such body tissues as muscles, tendons, blood vessels, fat, and connective tissues.

Certain cancers share some characteristics of soft-tissue sarcomas but are not placed in that group. These include most brain tumors and the so-called blood cancers, chiefly the leukemias. Some authorities include tumors of the lymph nodes--the lymphomas--with the soft-tissue sarcomas. The World Health Organization "International Classification of Tumors, No. 3, Histological Typing of Soft Tissue Tumors," however, excludes lymphomas and appears to be adequate for purposes of defining the malignancies in this category.

There is no evidence that all soft-tissue sarcomas have a common etiology or cause. These malignancies differ from one another as to how rapidly they grow and spread, how they are treated, and the results that treatment achieves.

These malignancies are rare. According to the National Cancer Institute, they comprise 2.76 percent of all cancer cases in men aged 25 to 29 and 0.58 percent of all cancer cases in men aged 55 to 59; the percentage declines because other types of cancers become increasingly common with age. Lymphomas, sometimes included with soft-tissue sarcomas, contribute another 5.21 percent at ages 25 to 29 and 2.40 percent at ages 55 to 59.

Although there is no evidence establishing a common cause for these sarcomas, some malignancies in the group are known to be associated with exposure to environmental hazards. For example, malignant mesothelioma is known to be caused by asbestos exposure, and angiosarcoma of the liver by exposure to vinyl chloride.

Because these malignancies are rare, it is difficult to devise adequate techniques to investigate their causes. A series of studies in Sweden using the "case/control" method grouped the soft-tissue sarcomas together in order to investigate whether Swedish foresters and farmers exposed to herbicides and a chemical known as chlorophenol in their work, suffered latent malignancies of this type. These studies have been carefully reviewed by Richard D. Remington, Dean of the School of Public Health, University of Michigan, at the request of the Office of Technology Assessment and determined to have been carefully conducted and well reported with results that suggest a relationship between herbicide exposure and soft-tissue sarcomas. Significantly, Dr. Remington pointed out the limitations of the case/control methodology and found the Swedish studies inadequate to permit definite conclusions.

Investigations in the United States based on studies of industrial workers have also suggested a phenoxy-compound connection with soft-tissue sarcomas. In addition, an East German investigation of malignant neoplasms among pesticide sprayers and agricultural technicians tends to support the Swedish studies by finding a single case of soft-tissue "malignancy," which probably was a soft-tissue sarcoma.

Other studies, in Finland, New Zealand, Great Britain, the Netherlands, and Italy have not confirmed the Swedish studies. In addition, a separate investigation of Swedish forestry workers casts some doubt on the Swedish studies.

Mr. Chairman, we do not disagree with Dr. Remington's conclusions as to the credibility and limitations of the Swedish studies. They lay a predicate for further investigation and do not rule out the possibility of a causal link. They do not, however, provide a reasonable basis upon which to favorably decide VA compensation claims.

We recognize the importance of careful scientific analysis in matters of this kind, and have attached to my prepared statement detailed background papers concerning these diseases.

The comprehensive epidemiological study mandated by Pub. L. No. 96-151, together with other ongoing studies including some devoted specifically to the soft-tissue sarcoma issue, may resolve many of the controversial questions raised by the use of Agent Orange in Vietnam. As I stated at the outset of my remarks, we must work toward the dual objectives of fair compensation for any Agent-Orange-caused disabilities and avoidance of steps that would compromise the integrity of the program. At this point, there is no evidence that either chloracne or PCT is a delayed effect of exposure, and we believe the provisions of H.R. 1961 respecting these disorders are not justified. We do not believe it has been satisfactorily demonstrated that exposure can cause soft-tissue sarcoma.

Accordingly, we oppose the enactment of H.R. 1961. In view of the current state of scientific findings, enactment would compromise the integrity of the compensation program and engender unfounded fears among Vietnam veterans that lethal illnesses may yet befall them as a result of having answered duty's call. Our biding moral obligation to veterans who have given so much demands that we act responsibly in all matters affecting the compensation program.

Mr. Chairman, that concludes my prepared remarks. My colleagues and I will be pleased to answer any questions.

## EPIDEMIOLOGY OF SOFT-TISSUE SARCOMA AND RELATED HUMAN RESEARCH

### Initiation of Swedish Studies of Herbicides and Cancer

In 1972 Swedish newspapers published rumors that railroad workers were dying from lung cancer as a result of exposure to herbicides used in their work. The National Board of Occupational Safety, as a result, requested Professor Olav Axelson, a specialist in occupational medicine, to undertake an epidemiological investigation of the matter.

The results of this investigation have been reported in a series of four papers, 1-4. Another series, 5-7, was prompted by criticism of the epidemiological and statistical methods employed in this and related studies. Attention in the United States has focussed on the pre-publication manuscript of the 1980 paper by Axelson, Sundell, Andersson, Edling, Hogstedt, and Kling, 4.

This paper dealt with two aspects of the study of railroad workers. The initial phase was a cohort study of 348 men who had been exposed, individually rather than as a group, to herbicides for more than 45 days during 1957 to 1972 and who were followed through October 1978. Exposure information was incomplete but the workers were divided into subcohorts with exposure to phenoxy acids (which include the ingredients of Agent Orange), amitrol, or to both herbicides. The mortality rates for these exposed subcohorts were compared to the age-specific national death rates for Swedish men, the latter serving as the control cohort.

Overall 49 deaths were expected in the exposed cohort; 45 occurred, a result attributed to the "healthy worker effect." There were, however, 17 tumors found where 11.85 were expected. Among the deaths occurring at least ten years after the first exposure, 6 cancers were found although only 1.78 were expected. Dr. Axelson, 8, later increased this to 7 tumor cases. Each subcohort had an excessive number of tumor deaths, the greatest being in the group exposed to both phenoxy herbicides and amitrol.

Although initially, 2, amitrol was associated with an increased tumor mortality, somewhat different results were found in a second phase of the examination, described as a case-referent study (identical to a case-control study). The data indicated a "statistical association" of phenoxy herbicides and excess tumor mortality, 3. Suspicion was increased by finding that workers exposed to phenoxy acids alone had a "statistically significant excess of stomach cancer", specifically 2 cases compared to 0.33 expected when this type of herbicide was used alone and 3 cases as compared to 5.1 expected (increased from 4.1, 8) for all workers exposed to phenoxy herbicides, alone or with amitrol.

The series of papers on railroad workers has been criticized on several methodological grounds, 9, and Axelson has replied to these criticisms, 6. Richard D. Remington, Dean of the School of Public Health, University of Michigan, reviewed this and other Swedish studies for the Office of Technology Assessment. His evaluation, 10, was that the Axelson investigations had been "carefully conducted" and "well reported." He pointed out the limitations of the statistical methods used and found that "the numbers available ... are inadequate to permit definite conclusions" although "the results ... are suggestive."

Of interest in connection with the question of soft-tissue sarcomas and phenoxy herbicides is the type of tumors found by Axelson's group, 4. One case of Hodgkin's lymphoma occurred among the eight tumors in men exposed to phenoxy herbicides alone and no soft-tissue sarcomas or non-Hodgkin's lymphomas was diagnosed among the eight tumors appearing in workers exposed to both amitrol and phenoxy herbicides. In other words, no sarcomas were reported for the total of 207 men exposed to phenoxy compounds, 2. A reticulum cell sarcoma and a Hodgkin's lymphoma were found among the 7 tumors of workers exposed to amitrol alone. Thus, there was one soft-tissue sarcoma reported for 152 men exposed to amitrol, 2. Another 28 persons described as exposed to "other herbicides and combinations" cannot be identified as to exposure to specific herbicides but apparently none developed a tumor.

Axelson's work is directly related to the later work on soft-tissue sarcomas and lymphomas by Lennart Hardell. Indeed, Axelson suggested to Hardell in 1976, that he conduct a case-control study of soft-tissue sarcomas and has actively assisted in Hardell's work since then.

## Swedish Investigation of Soft-Tissue Sarcoma

That work began when Hardell admitted for treatment 3 patients in the autumn of 1976, each with a soft-tissue sarcoma, and a history of exposure to phenoxy herbicides. He then found a total of 7 patients with "malignant mesenchymal tumors" (soft-tissue sarcomas) who gave a history of having worked with phenoxy herbicides 10 to 20 years earlier. The cases were among 87 patients with soft-tissue sarcomas, 55 of whom were men. Of these men, 9 were forestry workers, 6 worked in forestry and on farms, and 6 were employed in saw mills or pulp plants. Another two tumors appeared in men whose connection with forestry was less direct. The malignancies found were two leiomyosarcomas, two rhabdomyosarcomas, two neurofibrosarcomas, with one each of fibroid liposarcoma, myxofibrosarcoma, and polymorphocellular sarcoma, 12.

Following Axelson's advice, Hardell began a case-control study that was published in two journals, 13, 14, a common practice of reporting in Swedish with an almost identical paper in English. The second paper in English aroused much interest in the United States.

Hardell and Sandstrom found 21 living and 31 dead men who were diagnosed as having soft-tissue sarcomas in Hardell's oncology department in northern Sweden. They were matched for age and place of residence, as well as date of death for the deceased, with other men selected from the Swedish National Population Registry or from the National Registry for Causes of Death. Each living patient had 4 living controls; each dead man had 4 deceased controls. Exposure information was sought by the use of a mailed questionnaire that has never been published. It contained 130 questions, including 16 about the use of organic solvents, 4 about plastics, 3 about glues, 4 about drugs, "several" about smoking habits and an unstated number about exposure to phenoxy herbicides and chlorophenol as used in the lumber and paper mills. This questionnaire was mailed to the patient or to his next of kin if he were dead, 15.

When the answers to the questionnaire were less than clear, a supplementary interview was obtained, usually by telephone, with the interviewer unaware of the health status of the person in question. Employers, neighbors, and others were consulted "if necessary to verify and monitor the accuracy of the exposure information," 15.

Using the criteria for exposure established for the study, 36.5% of the 52 patients and 9.2% of the 208 controls had been exposed to phenoxy herbicides and/or chlorophenols. The "relative risk" of developing soft-tissue sarcomas was calculated as 5.7, i.e. men exposed to the chemicals had almost six times as great a change of developing a sarcoma as did those who were not exposed. The relative risk was 5.3 for the 46 men exposed to phenoxy herbicides alone, and 6.6 for the 40 men exposed only to chlorophenols. It was thought that confounding factors had an insignificant effect.

The authors concluded that "the investigation showed an increased risk for soft-tissue sarcomas" but "a specific evaluation of the effect of separate chemical substances was not possible," 14.

The study's methods have been criticized and doubts have been expressed about the 100% response rate to the questionnaire approach, 9. (Actually, 2 of 208 controls did not answer, 14.) The statistical approach was described as slightly misrepresented and a major criticism was leveled because of the possibility of "selective recall," the greater tendency for an ill person to remember a supposed "cause" for the illness than a well person would have to remember the same "causal" event.

The criticisms evoked several replies. Axelson defended the case-control design, the objectivity of obtaining exposure data retrospectively, and the statistical techniques, 6. He concluded that the use of interviews for determining exposure is justified, 7, and defended in principle the treatment of confounding factors, 16. Hardell recalculated the 1979 results and his subsequent papers to substantiate his earlier findings and performed a separate investigation in support of his confidence that "no substantial observational bias could exist in the studies," 15.

Remington, 10, expressed the view that "the findings of this particular investigation are suggestive" and that "a relative risk of 5.3 for exposure to phenoxyacetic acids must be taken seriously." However, "case-control studies are uniquely susceptible to hidden sources of bias" even when the investigators are "unusually careful" as they are in this "excellent investigation."

Hardell's group also undertook a second case-control study of identical design in southern Sweden which is more devoted to agriculture than to forestry, 17, 18. In this investigation each of 72 living and 38 dead patients was matched with two controls. Among the 110 cases, 22.7% reported exposure to phenoxy herbicides or chlorophenols and, among the 219 controls, 5.9% were so exposed. This gave a relative risk of 5.1 with matching and 4.7 when the matching was dissolved, i.e. when sorting by age was ignored during statistical calculations. The relative risk from exposure to phenoxy herbicides was calculated to be 6.8, and that from chlorophenols to be 3.3. Exposure to more than a dozen other noxious materials, e.g. asbestos, smoking, DDT, and lindane, were considered as possible confounding factors although none was found to be clearly associated with an increased risk by itself.

The reports list the diagnoses of all 110 cases of soft-tissue sarcoma as: leiomyosarcoma, 33; malignant fibrous histiocytoma, 19; liposarcoma, 15; neurogenic sarcoma, 11; angiosarcoma, 9; myxofibrosarcoma, 7; fibrosarcoma, 5; dermatofibrosarcoma, 3; atypical fibroxanthoma, synovial sarcoma, sarcoma NOS, 2 each; Ewings's sarcoma (extraskeletal) and rhabdomyosarcoma, 1 each. No statement is made as to which of these tumors was found in the 25 cases with identified exposures and no histological diagnoses are reported for the northern Swedish series, 13, 14.

The authors of the southern Swedish study conclude that "exposure to phenoxy acids and chlorophenols might constitute a risk factor in the development of soft tissue sarcomas," 18. The investigation has been the subject of the same criticisms and refutations as the earlier study.

Remington concludes that "the results are consistent with the hypothesis that phenoxy acid exposure increases the risk of tumors of this type" but adds that "case-control methodology is intrinsically susceptible to subtle and unmeasurable biases."

## Swedish Investigation of Lymphoma

In May, 1978, Hardell was prompted to a new study by a patient with a malignant histiocytic lymphoma and a history of "massive exposure to phenoxyacetic acids." All men admitted to the oncology department with this type of tumor during the first nine months of 1978 were asked about their occupation and possible chemical exposure. Of 17 patients, 14 reported an occupation consistent with exposure and 11 of them had had contact with phenoxy herbicides or chlorophenols ten or more years earlier, 19.

These observations led to a case-control study, the report of which in 1981, 21, differs considerably from that in 1980, 20. The earlier report was commented upon in manuscript form by various experts but the later version will be used here.

The investigation, in collaboration with Axelson, 20, included both Hodgkin's disease and non-Hodgkin lymphomas. The 169 cases consisted of 60 Hodgkin's disease patients (lymphocyte predominance, 20; nodular sclerosis, 3; mixed cellularity, 27; lymphocyte depletion, 10), 105 men with non-Hodgkin's lymphomas (follicular center cell (FCC) type, 53; non-FCC type, 52), and 4 individuals with unclassifiable lymphomas. Each case had two matched controls, 338 in all. Of the cases, 62 had died as had 124 of the controls.

Questionnaires and interviews were used to determine exposure to phenoxy herbicides, chlorophenols, organic solvents, or medicines and to characterize jobs, hobbies, and smoking as they were determined in the soft-tissue sarcoma investigations, 20. All cases and controls were from northern Sweden.

Cases in which exposure was reported to chlorophenol, or to "mutagenic" solvents (benzene, trichloroethylene, perchloroethylene and styrene) were divided into high-grade and low-grade exposure groups. Continuous exposure for a week or less or repeated exposures totaling less than a month were considered low-grade. Analyses also divided cases into two groups depending on whether 5 years had elapsed as a latency period between the first exposure to the chemical and the tumor diagnosis.

Of the cases, 36.1% had been exposed to phenoxy herbicides or chlorophenols; 9.6% of the controls had been so exposed. The relative risk for these exposures was 6.0 with matching and 5.3 without it. Phenoxy herbicides gave a relative risk of 4.8 although it was greater if exposure was for 90 days or more. Chlorophenols gave relative risks of 8.4 for high-grade exposure, 2.9 for low-grade. High- and low-grade exposure to organic solvents gave relative risks of 2.8 and 1.2 respectively. On the other hand the few cases with both phenoxy herbicide and high-grade organic solvent exposure was calculated to have a relative risk of 11.2 and some other combinations also gave large relative risks. The length of the latency period, however, seemed to have no effect.

The authors conclude that "this investigation suggests that exposure to organic solvents, chlorophenols, and/or phenoxy acids constitutes a risk factor for malignant lymphoma," 21. Dr. Remington commented that "a substantial and statistically significant relative risk is found for this group of tumors. And again, pnenoxy acid exposure is specifically incriminated." He continues, however, that the limitations of case-control methods have to be considered as well.

## Swedish Investigation of Carcinoma of the Colon

Hardell undertook to answer doubts that his questionnaire and interview methods allowed observational bias in assessing exposure by conducting a case-control study of "colon cancer." The condition is not suspected of having any association with phenoxy herbicides or chlorophenols. In consequence, if the previously used exposure determination resulted in a relative risk of 1.0 or near it, there had been no observational bias in the questionnaire-interview procedure used in the earlier studies of soft-tissue sarcomas and lymphomas.

Of the 157 men with colon cancer all but 3 answered the questionnaire. The controls consisted of the control groups from the soft-tissue sarcoma study (206 men) and the malignant lymphoma study (335 men). In all, 41% of the cases and 45% of the controls were dead. Of the cases and controls, 11.0 and 10.4% respectively had been exposed to phenoxy herbicides or chlorophenols. For phenoxy herbicides, the relative risk was calculated to be 1.3 and for chlorophenol it was 1.8. Neither was significantly above 1.0. The conclusion was that "the previously reported associations between exposure to phenoxy acids or chlorophenols and soft-tissue sarcoma and malignant lymphoma cannot to any essential degree be explained by observational bias," 15.

## Later Criticism of Swedish Studies

There remain, however, doubts about the practical significance of the Swedish epidemiological studies stemming from several of their characteristics. The main criticism is the reliance on recall of the men or their relatives, employers and associates for undramatic events years earlier as well as the possibility of unconscious bias on the part of the interviewer, the "observational bias" discussed above. Coggan and Acheson point out that the positive association between exposure to phenoxy herbicides and the development of several or many soft-tissue sarcomas, Hodgkin's disease and non-Hodgkin's lymphoma may indicate "a serious undetected bias" even though the explanation has been offered that all these tumors are embryologically related, 22. These authors conclude that "it is as yet impossible to estimate with any precision the risk of soft-tissue sarcoma due to phenoxy herbicides" but add that "there is suggestive evidence of a biological association between phenoxy herbicides (or their contaminants) and soft-tissue sarcomas." They feel that there is weaker evidence for an association between herbicides and lymphomas.

Hardell and Axelson reject the idea of observational bias, citing the colon cancer study as evidence, 23. They also defend the aggregation of tumors because of the "so-called addition theorum for chi-square and Poisson distributions" as well as the embryological relationship of the neoplastic tissues.

## American Support for Swedish Conclusions

Support for the connection between soft-tissue sarcomas and exposure to phenoxy compounds has been reported in several papers from outside Sweden. The data most often cited as favoring the relationship are derived from observations in the American chemical industry.* The first was a note by Honchar and Halperin in which they pointed out that of 105 deaths in four exposed industrial "cohorts" 3 (2.9%) were due to soft-tissue sarcoma, whereas only 0.07% of deaths among adult American men are so caused. The three cases were malignant fibrous histiocytoma, fibrosarcoma, and liposarcoma. The authors felt that these "suggest a common pattern," 24. Cook added a fourth case, another malignant fibrous histiocytoma and noted that all four were smokers and two had chloracne, 25.

Moses and Selikoff reported a fifth case, a non-smoker, with neurogenic sarcoma (malignant schwannoma). They give the total annual incidence of soft-tissue sarcomas as 4500 (less than 1% of newly diagnosed cancers) in the U.S. and quote 4.9% of soft-tissue sarcomas as malignant schwannoma, 26.

Johnson and his co-workers briefly described a young man who died of fibrosarcomatous mesothelioma some four years after first being exposed to phenol. His father had a liposarcoma after "prolonged exposure" in a plant manufacturing chlorinated phenols among other chemicals, 27.

Hardell and Ericksson accepted the two additional cases to total 7 deaths from soft-tissue sarcoma among 105 deaths among American industrial workers, the expected number being 0.07%. This would "fit in with" the Swedish investigations, they believe, 28.

To date no critical review has been made of the cases and the industrial population in which they were detected. The reports have been brief "Letters to the Editor" and each discusses one to three cases. The total of 105 deaths used as the number of dead workers has not been kept current as new soft-tissue sarcoma cases were added and the total number of exposed workers has not been given. No use has been made of controls, even in the form of a retrospective cohort comparison.

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*Data given by Honchar and Halperin, Cook, Moses and Selikoff, and Johnson et al pertain to workers at Monsanto Company and Dow Chmical Company. For details of studies of these workers see 24a and 25a.

A case report without statistical data briefly described three soft-tissue sarcomas among Vietnam veterans who reported exposure to phenoxy herbicides in that country. One man had an inflammatory histiocytoma, another suffered from a fibrosarcoma, and the third had a leiomyosarcoma, 29.

## European Support for Swedish Conclusions

Barthel determined the frequency of malignant neoplasms among 1791 pesticide sprayers and agricultural technicians in East Germany during 1976 to 1979. He states the retrospective cohort study used police as controls but gives no data for them. After eliminating "on statistical grounds" 133 cases who died before 1970, he compared the mortality rate and cancer incidence with corresponding figures from the death statistics and the cancer registry of the Health and Social Welfare. The "case" group had multiple exposures over the years to fungicides, insecticides, and herbicides including phenocyacetic acids. Among 169 malignant neoplasms in 1658 exposed men were 1 lymphosarcoma, 3 plasmacytomas, 1 described as a malignancy of lymphoid tissue, and 1 of soft-tissue, not otherwise characterized. Bronchogenic carcinoma was the most common malignancy with 59 cases, double the expected occurrence, although the cases had smoking habits like those of the general population, 30. A brief report describes a case of non-Hodgkin's lymphoma and a second of malignant lymphoma among 158 workers with pentachlorophenol. This type of neoplasm would have an expected occurance of 0.28, 31.

## Studies Not Supporting Swedish Conclusions

In contrast to the reports of an association between phenoxy herbicides or related compounds and soft-tissue sarcomas and malignant lymphomas, some investigators have found no association. Some of these investigated a possible relation, others were "follow-up" studies of industrial workers in whom no sarcomas or lymphomas were found.

Dr. Riikimaki and his collaborators have completed nine years of mortality study following 1,926 persons who worked with phenoxy herbicides in Finland during the 1955-1971 period. All had at least two weeks of exposure and a quarter of the men totalled eight weeks or more as of 1971. The mortality rates among the workers were compared with the national death rates. As of 1980, there had been 82 deaths of exposed men as compared to 91 expected and, of these, 17 were cancer deaths with 18.4 expected. There were no cases of soft-tissue sarcoma nor of lymphomas although 0.1 and 0.8 would have been expected. The authors believe that "the investigation cannot be regarded as a conclusive negative study" but point out that the "results do not confirm the ... association between mixed herbicide exposures" and cancer risk, 32.

Hogstedt and Westerlund compared the mortality rate of Swedish supervisors and workers in forestry. The supervisors were fewer in number (142) than the workers (244) but the former were judged to have been more heavily exposed. The relative risk of death was about the expected but, after a 10-year latent period, the relative risk for cancer was about 4 for the supervisors and only about 0.4 for the workers. The fatal tumors were of various types but there was no soft-tissue sarcoma or lymphoma, 33.

Two case-control studies in New Zealand have been initiated by Smith et al to examine the association suggested by Swedish studies of phenoxy herbicides with soft-tissue sarcomas and malignant lymphomas. In the first investigation, 102 cases of soft-tissue sarcoma have been identified in men from the New Zealand Cancer Registry between 1976 and 1980. An equal number of matched controls with other forms of cancer were selected for comparison. The sarcomas are fibrosarcomas, 25; liposarcomas, 20; rhabdomyosarcomas, 9; leiomyosarcomas, 7; malignant histiocytomas, 6; other types, 22; and unspecified, 13. The preliminary report compares cases and controls as to the occupation shown on the Registry enrollment. There was no significant difference betwen the groups as to the number of men working in agriculture, forestry, and fishing, the occupations with the greatest likelihood of exposure to phenoxy herbicides and chlorophenols. The only occupations associated with soft-tissue sarcomas exclusively are blacksmiths, machine tool operators, electrical fitters, and electrical workers. The investigators are now obtaining work histories for cases and controls by telephone interviews and warn that later results may change their conclusions. The data at present "do not give evidence for a relationship (of soft-tissue sarcoma) with occupational exposure to phenoxy herbicides and chlorophenols" but "should not be taken as substantive evidence against the hypothesis", 34.

A second report by Smith et al includes the results of the telephone interviews regarding 80 cases and 92 controls already completed. Probable or definite exposure to phenoxy herbicides for more than one day earlier than five years before cancer registration was found in 17 cases and 13 controls, giving an odds ratio of 1.6. This would be expected to increase when the exposure criteria were more stringent, but, when exposure was at least five days and more than ten years before registration, there were 13 cases and 12 controls included reducing odds ratio of 1.3. Neither ratio is statistically significant and there have been no soft-tissue sarcomas reported among the most highly exposed group of 2000 aerial and ground sprayers. The results, the authors believe, "do not generally support the hypothesis that exposure to phenoxy acid herbicides cause soft-tissue sarcoma," 35.

A brief initial report by Edling and Granstam compared the causes of death for 375 Swedish forestry workers, aged 25 to 69 years, who died during 1968 to 1977, with the mortality figures from the Swedish national statistics. There were 75 deaths from all malignant tumors, as compared to 86 expected. Renal tumors killed 8 with 3.84 expected and "tumors of lymphatic and hematopoetic systems" were responsible for 14 deaths with 7.5 expected. No deaths were attributed to soft-tissue sarcoma, 36.

In addition to these studies, several small industrial groups have been followed well into the latent period for solid tumors. None has been reported to include cases of soft-tissue sarcoma or malignant lymphoma. May examined 41 of 79 workers who developed chloracne following accidental exposure to trichlorophenol in 1968 at the Coalite Company in Great Britain. Another 54 employees were possibly exposed. None of the workers had significant changes ten years after the accident and neither death from nor evidence of neoplasm was found, 37. Jirasek's group has closely followed 55 men who were intensely exposed during the manufacture of 2,4,5-trichlorophenoxyacetate from 1965 to 1968 in Spolana, Czechoslovakia, and who developed evidence of acute intoxication. Two workers died of bronchogenic carcinoma 5 to 5.5 years after the first exposure. There was no other evidence of malignant neoplasms during a ten-year follow-up, 38.

In 1963 an explosion at Philips-Duphar, Amsterdam, exposed 106 workers involved in manufacturing 2,4,5-tetrachlorophenyoxy acewtate. Among the 93 workers followed to 1977, only one death 14 months after the accident was due to cancer and the pancreatic carcinoma involved was apparently symtomatic before the explosion. No case of soft-tissue sarcoma or malignant lymphoma was reported, 39.

One study is often cited with the Swedish studies although it did not deal with soft-tissue sarcomas and malignant lymphomas, 40. A more recent review by Thiess et al reports that all 74 exposed persons are still being followed after 26 years. There have been 21 deaths, about equal to the 18 to 20 deaths expected from major comparative populations and 18 and 19 deaths expected among matched unexposed controls. Cancer was responsible for 7 deaths as compared to 4.1 expected from the comparative populations and 5 in each internal control group. Gastric carcinoma in 3 exposed persons exceeds the expected 0.61 to 0.70 expected cases. There were, however, no soft-tissue sarcomas or malignant lymphomas among these chemical workers at BASF, 41.

A number of other industrial exposures to phenoxy herbicides, their precursors or contaminants were reported before 1973, 42. The populations were small but generally heavily exposed. Unfortunately it has not been possible to locate late reports on the exposed populations although ten years or more have elapsed since exposure.

The accident at the ICMESA factory in Seveso, Italy, in July 1976 exposed many people to trichlorphenol; more than 5400 adults and children of both sexes are known to have been in contact with the chemicals for several days, 43. Although only about six years have elapsed since the exposure, the population has been under surveillance and the rate and causes of death are being followed. To date no soft-tissue sarcomas or malignant lymphomas have been reported.

Another less systematic observation bears on the situation. The phenoxy herbicides have been used frequently and extensively in agriculture and forestry in the United States since the late 1940's. They were used on lawns in cities, as well, for most of that period. If the relative risk of developing so distinctive a group of tumors as the soft-tissue sarcomas and the malignant lymphomas had increased by 5 of 6 fold over that before 1945 as the Swedish studies would predict, it almost certainly would have been evident to clinicians and pathologists, especially in the rural areas, even without systematic studies. No such increase was noted.

## Critical Evaluations

The Swedish investigators have been cautious in interpreting their results. In his medical dissertation based on his epidemiological studies, Hardell judges that the similar results in the two case-control investigations (12, 13, 14, 16) "seem to increase the confidence that the observed association of exposure to phenoxy acids and soft-tissue sarcoma was not spurious" and did not believe that confounding factors "could account for the observed relation." In summary, he concluded that "it is suggested that exposure to phenoxy acids should be looked upon as an occupational cancer hazard," 44.

Other reviewers have been more skeptical as to the significance of the work. Remington's overall opinion was that "in toto, the Swedish work is credible if not fully conclusive. Certainly this work would seem to justify further investigation," 10. Coggan and Acheson, after reviewing other work as well as the Swedish studies, state that "on the present evidence it seems possible that soft-tissue sarcomas have arisen in association with exposure to phenoxy herbicides" but continue that "it is as yet impossible to estimate with any precision the risk of soft-tissue sarcoma due to phenoxy herbicides." They conclude that "there is suggestive evidence of a biological association between phenoxy herbicides (or their contaminants) and soft-tissue sarcoma. The evidence relating these products to the occurance of lymphoma is weaker," 22. An unsigned editorial in Lancet commenting on the opinions of Coggan and Acheson seems to agree with their conclusions with regard to soft-tissue sarcomas, 45.

Hardell and Axelson disagreed with both the Coggan and Acheson's opinions and the Lancet editorial, 23. They have been at some pains to counter charges of "observational bias," 15, but have not convinced everyone that faulty memories do not result in significant errors in evaluating exposure, 45.

The causal connection between phenoxy herbicides and soft-tissue sarcomas would be much more likely if there were a unique preponderance of one type or even of a few types in the exposed men. The Swedish reports never compare the morphological types or location of the malignant tumors in cases with those in controls, 45. Their only justification for aggregating the types, and presumably for omitting the data from their reports is "the uncertainty of relations between the various histological groups in terms of causal mechanisms" and "the so-called addition theorem for chi-square and Poisson distribution," 23. The uncertainty of causal relations is precisely the reason for reporting the groups and the addition theorem cannot justify the aggregation of unlike

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*2,3,7,8-tetrachlorodibenzo-p-dioxin has been suggested as the principal carcinogen in the phenoxy herbicide 2,4,5-T and trichlorophenols but this has been disputed. See 22, 23, 45, 46. The controversy is not considered in this discussion.

entities unless significant common factors have been demonstrated.

Scientific results are strengthened greatly when independent investigators substantiate them. The Swedish studies have been said to be independent and confirmatory. The two soft-tissue sarcoma investigations do support one another (12, 13, 14, 16) but they are the work of the same group of investigators. The investigation of malignant histiocytic lymphoma was also conducted by the same group but was a case-control study of a separate entity, (19-21). Axelson's work on herbicide exposure and cancer (1-4) was not truly independent from Hardell's efforts since Hardell has recognized his indebtedness to Axelson for his assistance in the first case-control study, (13, 14). More important Axelson did not associate phenoxy herbicides or chlorophenolic compounds with soft-tissue sarcomas nor with malignant lymphomas among railroad workers, 1-4.

The reports of soft-tissue sarcomas among chlorphenol workers in the United States (24-27) have been cited as supporting Hardell's conclusions, 24, 28, 44, 46. The data have been reported piecemeal without a clearly enumerated total population from which they were drawn. The comparison was made to mortality data for the general population of the appropriate age and sex. The type of soft-tissue sarcoma is known for each case; among the 7 men were 2 malignant fibrous histiocytomas, 2 liposarcomas, as well as one each of fibrosarcoma, malignant schwannoma, and fibrosarcomatous mesothelioma. As before, the tumors are not of a uniform type.

Coggan and Acheson comment that the Swedish studies and the American reports taken separately do not "provide convincing evidence that the incidence of soft-tissue sarcomas is increased after exposure to phenoxy acids and chlorophenols, -- Considered together the whole becomes more persuasive." They add that "it is surprising that the association should apply to tumors of such a variety of tissues," 22. The *Lancet* editorial finds only that "the number of deaths due to soft-tissue sarcomas [in the American data] is disturbing," 45.

In addition to the American experience, the British (37), European (30, 32, 33, 36, 38-41) and New Zealand (34, 35) medical and scientific writers have studied populations five years or longer after exposure to phenoxy herbicides and/or chlorophenols in a variety of situations, some intense and acute, others prolonged. Only one observer (30) reported a case described as a soft-tissue malignant neoplasm without further characterization. The same report included a lymphosarcoma, a malignant neoplasm of lymphoid tissue and 3 plasmocytomas. No other study found a soft-tissue tumor.

In summary, the Swedish studies of soft-tissue sarcomas cannot be considered to have proved that exposure to phenoxy herbicides is the cause of one or more types of this varied group of malignant tumors. There are no fully reported systematic studies to confirm what the Swedish investigators describe as an association. There are an epidemiological study (32) and observations of exposed populations that do not support the finding as opposed to uncorrelated American observations and an East German study (30) that do strengthen the case for such an association.

At best, the Scottish verdict of "Not proven" seems most realistic at this time. The Advisory Panel on Toxic Substances of the American Medical Association says that "while 2,4,5-T and 2,4-D pesticides (phenoxy herbicides in Agent Orange) have been used in agriculture, forest management and residential landscaping for over 30 years, there is still no conclusive evidence that they and/or TCDD (a contaminant of Agent Orange) are mutagenic, carcinogenic, or teratogenic in man, nor that they have caused reproductive difficulties in the human," 47.

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## PORPHYRIA CUTANEA TARDA

Definition: Porphyria cutanea tarda (PCT) is the most frequent of a group of uncommon diseases, the porphyrias, that comprise disturbances in the body's formation of hemoglobin, the red chemical in blood. A specific pattern of chemicals called porphyrins, excreted in the urine and stool, characterizes PCT and reflects a deficiency of one of the liver's enzymes involved in hemoglobin formation. The disease manifests itself in the skin where small and large blisters form on the exposed parts of the body, probably as a slow response to sunlight. The skin becomes very fragile and easily rubs off to produce sores that scab over and sometimes leave scars. PCT appears to be inherited, at least in some cases, but exposure to some chemical or other external factor is required before it becomes manifest. (1-4)

Clinical Manifestations: PCT is usually first noticed when small or large blisters appear on the face, the back of the hands, and the arms usually following exposure to sunlight many hours or days earlier. This contrasts with the prompt reaction to sunlight in other porphyrias. The skin slowly becomes so fragile that even slight rubbing strips off the top layer leaving an open shallow sore that scabs over and heals slowly. The thin skin that forms in the area gradually returns to normal over months. It may instead scar permanently or, in severe cases, may progressively stiffen and thicken until it resembles another skin disease, scleroderma. (5, 6) In a few very severe cases, the skin changes have resulted in the loss of parts of the nose, ears or fingers. (2)

Hair, especially around the temples and upper cheeks, may grow dark and prominent. (7) The skin may darken or may lighten abnormally in the affected areas. (8) Small "white heads" sometimes appear before or after blistering.

The porphyrins in the urine often give it a pink, dark red or brown color. They fluoresce red in ultra-violet light, a phenomenon that assists in diagnosing PCT. (3)

Several methods of treating PCT are available, the most frequently used being repeated bleeding, and the disease is more successfully treated than are other porphyrias. The most important measure, however, is the avoidance of any chemical or other factor that precipitated the attack. (1, 2)

The time it takes the patient to recover seems to depend upon how severely the body was damaged. The skin changes disappear first, usually within 6 months when even severe cases are treated. (9, 10) The disturbance of the body's chemistry clears up in about a year. In less severe cases, simply avoiding contact with the causative chemical or other external factor is followed by complete recovery within a year. (11) Children who developed PCT after prolonged and intensive exposure when they ate seed grain treated with a chemical were very ill and had scarring, hairiness, arthritis and stunted growth even 20 years later. (12)

PCT is usually accompanied by some liver damage and in 4 percent or more of the patients a particular form of liver cancer develops. (1) It is difficult to determine whether the liver is damaged by PCT or by alcoholism which often precipitates the porphyria.

Causes: At present, PCT is thought to have two causes: heredity and external factors. There is little agreement on the relative role of these two or on exactly how they interact.

Heredity: There is good evidence that some cases of PCT have a hereditary basis. This has been demonstrated by finding, in healthy blood relatives, the same chemical defects as appear in patients although the changes are less severe in the well relatives. (13 - 16) The few available studies (16, 17) indicate that 6 to 10 percent of the general population have the hereditary defect; most never develop obvious PCT.

The various family studies show that PCT is inherited as what geneticists call an "autosomal dominant gene." (13 - 16) This gene decreases the liver's ability to produce an enzyme called "uroporphyrinogen decarboxylase" that is essential for the normal production of hemoglobin. A deficiency of this enzyme can result in the appearance in the urine of unusually large amounts of substances called "uroporphyrins" as well as related changes in the body's chemistry. (1 - 4)

The situation is more complicated, however, than this suggests. Not all persons who have the hereditary defect develop PCT and not all persons with PCT have evidence of the hereditary defect. It apparently requires exposure to some other factor, generally an environmental one, to produce the disease prophyria cutanea tarda even in the presence of the hereditary defect.

External Factors: A wide variety of chemicals, diseases, and even bodily states can disturb the liver's ability to bring about normal chemical reactions in the formation of hemoglobin and so produce one or another type of prophyria. (18) Some of these changes are relatively slight and transient; others are more serious and longer lasting.

The chemicals that produce PCT have been divided by some investigators into two catagories: those that trigger attacks in people with the hereditary enzyme defect and those that produce the disease in people without the defect. The chemical hexachlorobenzene is most often given as an example of a substance that produces PCT in anyone. This is largely because 4000 people in rural Turkey who ate, over several winters, seed grain treated with the chemical developed attacks of PCT that were often severe and protracted. (12) Relatively large doses of the female hormone estrogen is said to produce PCT only when the hereditary defect is present. (2). The interaction of hereditary and external factors is still not understood but it is important in several situations, two of which are alcoholism and exposure to TCDD or dioxin.

Chronic alcoholism is unquestionably the most common precipitating factor producing PCT among the populations of North America and Europe where about 68 percent of PCT patients are alcoholic. (19) The alcoholic patients who develop PCT, however, have often not been so impaired by their drinking that they could not hold jobs.

Alcohol abuse has many deleterious effects, of course. Among them are the storage of an excessive amount of iron in the liver and the production of various liver changes culminating in hepatic cirrhosis. Both of these changes are associated with PCT but exactly how they are related to it is not clear. (1) While alcoholism is common among PCT patients only about 2 percent of alcoholic patients with cirrhosis develop uroporphyrinogen decarboxylase deficiency and PCT. (20) This suggests that a hereditary defect probably plays a part in the appearance of PCT in alcoholics.

TCDD or dioxin (2,3,7,8-tetrachlorodibenzo-p-dioxin) has been implicated as the cause of PCT in only two industrial episodes, both involving prolonged contact with large amounts of the chemical. In both instances, the workers were exposed to other chemicals as well.

At the Diamond Chemical plant in New Jersey that manufactured chemicals containing TCDD, 55 men were examined in 1963 when 3 workers were found to have had the skin and hair changes of PCT and to pass urine containing uroporphyrin. When no longer in contact with TCDD, one man recovered completely within a year and another had recovered during a two-year period. The third had only some scars a year after being removed from contact with TCDD. Another 11 men had uroporphyrin in their urine without skin changes. Of the 55 men, 17 had the other skin disease, chloracne, indicating exposure to TCDD or a related chemical but there was no relationship between the occurrence of PCT and of chloracne. (21)

Six years after the initial examination, a second group of doctors examined 73 men working at the same New Jersey plant which had taken steps to protect its employees from exposure to TCDD after the earlier episode. No PCT was found and only one worker continued to pass uroporphyrin. Some men still had slight to moderate chloracne, however, (22)

The second industrial episode involved intense exposure to similar chemicals, including TCDD, between 1965 and 1968 in a manufacturing plant in Czechoslovakia. In all, 80 of 400 workers became ill. (23) A total of 78 developed chloracne as evidence of contact with TCDD or pentachlorophenol both of which were present. Twelve workers, more than half of them over 40 years of age, were diagnosed as having PCT. One man is said to have been exposed to the chemicals for only two and a half weeks before he developed PCT and in one patient porphyria is reported to have progressed rapidly into hardening of his brain's arteries. (24) Neither the rapid onset nor the progression to arterial hardening is known to occur in PCT.

Among 55 workers examined repeatedly, 11 persistently had large amounts of uroporphyrin in the urine; 12 others intermittently passed large amounts. These values gradually decreased during a four-year period. Of the 11 workers with a heavy output of porphyrin, 10 had the usual skin changes of PCT. (25)

The patients knew of no porphyria in members of their families and the researchers were unable to determine whether any of the men drank excessively. (26) In 1974, the doctors reported that the amount of uroporphyrin in the urine had been greatest in 1969 but had returned to normal as the skin changes improved. After 9 years of observation excessive excretion of uroporphyrin and skin manifestations were "exceptional" occurrences. (27) A year later such abnormalities were said to be "very rare." (23)

In addition to these two incidents of PCT as a consequence of exposure to high concentrations of TCDD, other researchers have described a less severe and completely non-symptomatic change in the liver's enzyme performance following the industrial accident at Seveso, Italy in 1976. Two years after the accident the amount of porphyrins in the urine was normal but the types excreted showed minimal changes in 84% of the people examined. This may indicate that TCDD has an effect but it did not indicate that the people had any disease. (28)

To make matters more complicated two related persons who lived near Seveso were found to have PCT. Examination of 66 family members demonstrated, however, that the two were suffering from the hereditary form of PCT. (29) It raises the possibility that TCDD as an environmental factor may have enabled the hereditary enzyme defect to exert its adverse effect.

Treatment: The most important therapeutic and preventive measure for PCT patients is the avoidance so far as possible of further contact with the external factor or factors that precipitate the attacks. This is critical whether alcohol, estrogen, polychlorinated biphenyls (PCBs), hexachlorobenzene, certain drugs, TCDD, or other factors are involved.

The usual active treatment is repeated bleeding. In most patients with severe PCT there are excessive iron stores, especially in the liver. Judicious bleedings, as in blood donations, remove this iron and improve the patient's condition. There are medications that can be used to reduce the body's iron stores although generally they are not as safe as bleeding.

Untreated, the disease grows worse year by year if the external factors are not removed. Eventually the liver becomes seriously damaged. Removal of the external cause, however, especially after a single or a few relatively brief contacts, is followed by slow but progressive improvement with apparent recovery. (30)

Agent Orange as External Factor: Since one component of Agent Orange contained small amounts of TCDD, it has been suggested that the herbicide acted as an external factor to cause porphyria cutanea tarda, with persistence of the condition to produce continuing trouble at the present time. This seems unlikely for several reasons.

PCT is a skin problem with a dramatic appearance and under conditions of combat in Vietnam would probably have been incapacitating, unlike chloracne which would have been relatively inconspicuous in most cases. In New Jersey and Czechoslovakia, the only two episodes where TCDD is known to have produced PCT, the skin changes were readily noticed.

The industrial exposures to TCDD causing PCT were intense and prolonged, lasting several years for most of the workers. Troops in Vietnam were exposed to much less TCDD.

Recovery from PCT usually follows removal from contact with the external cause within five years. The last contact with Agent Orange in Vietnam was ten years ago.

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# DIOXIN AND REPRODUCTIVE EVENTS

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Polychlorinated dibenzodioxins, sometimes referred to as PCDD's or simply dioxins, may be formed during the combustion of fossil fuels, the incineration of municipal wastes, or the production of chlorinated phenols.[1] Of all of the isomers of dioxins, 2,3,7,8-tetrachlorodibenzodioxin or TCDD, a low level contaminant of 2,4,5-T, trichlorophenol and its by-products, is considered the most toxic.[2]

A variety of biological effects have been indicated or hypothesized to be associated with exposures to TCDD. One set involves the potential influence of this chemical on reproduction. Animal experiments suggest adverse reproductive events may be associated with maternal exposures, but not with those of the father.[3,4,5] Reports of observational studies of humans are somewhat conflicting. The National Academy of Sciences, commissioned to study the effects of herbicides in South Vietnam, found no conclusive evidence of an association between exposure to herbicides and birth defects in humans.[6] Field and Kerr, in an ecological study in Australia, purported to have found a "linear correlation" between neural tube defects and increased use of the herbicide 2,4,5-trichlorophenoxy acetic acid (2,4,5-T), an agricultural chemical contaminated by low levels of TCDD.[7] On the other hand, Thomas reported that in Hungary, where 2,4,5-T usage had increased dramatically between 1970 and 1976, the frequency of stillbirths and congenital malformations had remained stable or decreased.[8] Smith recently published a "preliminary report of reproductive outcomes among pesticide applicators using 2,4,5-T in New Zealand".[9] No significant differences were found in the rates of congenital defects, stillbirths, and miscarriages between the chemical applicators and a control group.

There have also been dioxin-related claims of congenital malformations among offspring of United States and Vietnamese veterans of the Vietnam conflict who were exposed to aerial herbicides sprayings;[10,11] and of increased spontaneous abortions among women residing in or near areas of Oregon sprayed with 2,4,5-T.[12] None of these allegations have been substantiated epidemiologically and the Oregon spontaneous abortion data, when subjected to critical review, were found not to support the conclusion of an association with herbicide usage.[13]

Dow Chemical USA has conducted a series of epidemiological studies of employees potentially exposed to dioxin.[14,15] The following is a summary of a paper by Townsend et al, which has been submitted for publication.[16] It describes the results of an interviewer-administered questionnaire survey conducted among women whose husbands were employees of the Michigan Division of Dow Chemical U.S.A. The purpose of the survey was to compare the proportion of adverse outcomes between pregnancies associated with potential paternal exposures to 2,3,7,8-TCDD or other polychlorinated dioxins, and pregnancies with no known exposures to these chemicals.

## MATERIALS AND METHODS

### Selection of Study Subjects

Identification of wives whose husbands had been potentially exposed to dioxins, and of those whose husbands had not, was accomplished via company records. Using annual personnel census lists and individual work histories, employees who had been assigned to chlorophenol processes for at least one month between January 1939 and December 1975 were identified. By this criterion, there were 930 male employees with potential occupational dioxin exposures.

A comparable number of controls was selected from male employees who had no job assignments in the potential exposure areas, and who closely matched exposed employees by date of hire. By a variety of methods, addresses were obtained on all but 69 (3.7 percent) of the 1830 men. For the purposes of study efficiency, followup was limited to those who lived in Midland or adjacent counties. In the exposed group, 586 had wives potentially available for interview and 370 or 63% agreed to participate. Among the control group, 559 wives were potentially available and 345 or 62% were interviewed.

### Estimation of Exposures

Manufacture of pentachlorophenol started at the Michigan Division of Dow Chemical, U.S.A. in 1939. Trichlorophenol production started in 1946. In late 1963, a change was made in the trichlorophenol process and shortly thereafter a number of employees contracted chloracne. Operations were modified to reduce exposures and, in 1966, trichlorophenol production was transferred to a new building.

Estimation of exposures was made by an industrial hygienist who was familiar with the processes. Based on his experience, information provided by production personnel and surface contamination data, each job was assigned an exposure potential on a scale of low to high for TCDD, for all other dioxins, or for mixed exposures.

## Collection and Analysis of Data

A letter explaining the purpose of the study and requesting a personal interview was mailed to the residence of each living exposed or control population member residing in Midland or adjacent counties. Appointments were made with those women willing to be interviewed. If the employee was widowed or single, or if the response indicated the wife did not wish to be interviewed, no further action was taken.

Interviews were conducted in the home by one of three trained nurse-interviewers. Appointments were arranged by geographic clusters and not by exposure status. The interviewers did not know whether they were interviewing the wives of exposed or control empolyees. At the time of the interview, the intent of the study was re-explained, each woman was advised regarding confidentiality of the information and was asked to sign an informed consent. A five-page questionnaire was used and the interview normally took less than one half-hour.

Responses dealing with health defects, congential malformations and infant deaths were coded by the Eighth Revision of the International Classification of Diseases (ICD-8).[9] Each positive response regarding congenital malformations was coded if the mother stated the condition was diagnosed prior to one year after birth or if the mother was unclear as to the time of diagnosis. Conditions identified after the first birthday were reviewed by two physicians. If either physician, blinded as to the mother's group, determined the defect could have been diagnosed during the first year of life, the condition was so coded. Since these procedures were weighted toward identifying abnormalities, the resulting data could not be meaningfully compared with state or national statistics.

For the main purposes of this study, the individual conceptus or product of conception was the observational unit, taken as independent of other conceptuses of the same pregnancy or other pregnancies of the same or different womrn. Effect of exposure to dioxins was considered to be an irreparable event; that is, any exposure of a male employee any time prior to the estimated date of each conception attributed to him was investigated as an independent variable with potential effects on the pregnancy outcome. Conception dates were estimated from the length of pregnancy and pregnancy outcome dates reported in the questionnaire. When the date of conception preceded the father's first exposure, the conceptus was assigned to the no-exposure category even though the father was in the dioxins group. This prevented a dilutional effect on the risk estimates, while simultaneously making maximum use of the available data. Two new files were thereby created, one containing only "dioxins" conceptuses and the other "non-dioxins" conceptuses.

## Variables

Dependent variables included live births and the adverse outcomes of miscarriages, stillbirths, infant deaths, and live births with congenital malformations. Birth defects were analyzed as either health defects, congenital malformations, or as indicator malformations. A health defect was defined as a live birth reported by the mother to have a health problem of any nature. A congenital malformation was a report by the mother of one or more defects in the 740-759.9 range of ICD-8.[17] And an indicator malformation, one that we felt would be readily observed by the mother, was any one of the following (preceded by its ICD-8 code):

| | |
|---|---|
| 741.0 | Spina Bifida |
| 742.0 | Congenital Hydrocephalus |
| 749.0 | Cleft Palate |
| 749.1 | Cleft Lip |
| 749.2 | Cleft Palate with Cleft Lip |
| 755.0 | Ploydactyly |
| 755.1 | Syndactyly, and |
| 759.3 | Down's Syndrome |

Thus, indicator malformations were a subset of congenital malformations which, in turn, constitute a subset of health defects.

Independent variables consisted of four exposure categories:

TCDD Only, at any level
Any Dioxins (TCDD and others), at any level
Moderate to High TCDD Only, and
Other Dioxins Only (no TCDD).

The first two were also broken down into two duration-of-exposure subcategories: equal to or less than one year, and greater than one year total exposure.

Covariables were nine potential confounding factors or other reasons for adverse reproductive outcomes referenced in the literature: age, birth control method, complications during labor and delivery, conditions during pregnancy, medications or treatment during pregnancy, alcohol consumption, gravidity, high risk job, and smoking during pregnancy (Table 1). You will note from the Table, many were broken down into subcategories. Complications during labor and delivery were only considered a potential confounder for the analysis of stillbirths. High risk jobs were those held by the mother during pregnancy, such as operating room technician, x-ray technician, bacteriologist, cosmetologist, and dental assistant. Smoking was confined to that pregnancy, and not life-time experience.

## Statistical Analysis

Statistical analyses attempted to determine whether there were differences in the distribution of outcomes between the "exposed" and "non-exposed" conceptuses. This was done on crude or unadjusted outcomes, and after adjusting by stratification for the influences of pertinent covariables. Trend analyses were also examined for the exposures to TCDD Alone or to Dioxins Of Any Type, that is, whether there were trends of increasing adverse pregnancy outcomes with increasing durations of exposure.

## RESULTS

Interviews were obtained from 370 wives of potentially exposed and 345 wives of control employees (Table 2). Eleven in the first group and 13 in the second reported no pregnancies, 3.0 and 3.8 percent, respectively. A total of 1503 conceptions occurred among wives of the exposed and 1274 among those of the unexposed, or 4.06 and 3.69 conceptions per woman, exposed and unexposed.

As mentioned earlier, most of the analyses used the conceptus as the observational unit. Therefore, those conceptuses conceived by an "exposed" employee prior to his exposure to dioxin were considered to be "non-exposed" conceptuses and placed in the control pool for the purposes of the analyses. As illustrated diagrammatically in Figure 1, the consequence of this strategy was an increase in the number of "non-exposed" and a corresponding decrease in the number of "exposed" conceptuses. The 370 wives of employees exposed some time between 1939 and 1975, had a total of 1503 conceptions, but only 737 occurred after their husbands' first exposure to dioxin. These 737 constitute the "exposed" conceptuses. Thus, 1503 minus 737 or 766 conceptions occurred before the father was employed in a dioxins area.

We had two options with regard to the data on the 766. We could throw the data away, or add the experience of those 766 "pre-exposure" conceptuses to the 1274 conceptuses that occurred among wives of never exposed employees. We chose the latter. That gave us 766 plus 1274 or a grand total of 2040 conceptuses in the "unexposed" category. There were 9 induced abortions among this latter group, which reduced the number to 2031. The rest of the discussion

Table 1. Covariables

| |
|---|
| Age |
| <18, 18-30, >30 Years |
| Birth Control |
| None, Pills, IUD, Other |
| Complications During Labor and Delivery |
| Conditions During Pregnancy |
| Medications |
| Alcohol |
| ≤6 or >6 Drinks Per Month |
| Gravidity |
| 1, 2 or 3, > 3 |
| High Risk Job |
| Smoking |

Table 2. Summary of Pregnancies

| | Exposed | Unexposed |
|---|---|---|
| Interviews | 370 | 345 |
| No Pregnancies | | |
| Number | 11 | 13 |
| Percent | 3.0 | 3.8 |
| Conceptions | | |
| Number | 1503 | 1274 |
| Mean | 4.06 | 3.69 |

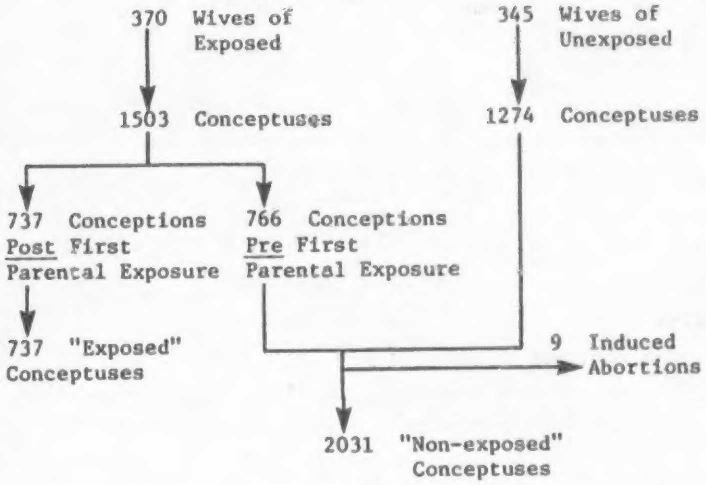
370 Wives of Exposed
345 Wives of Unexposed
1503 Conceptuses
1274 Conceptuses
737 Conceptions Post First Parental Exposure
766 Conceptions Pre First Parental Exposure
737 "Exposed" Conceptuses
9 Induced Abortions
2031 "Non-exposed" Conceptuses

Figure 1. "Exposed" and "Non-exposed" Conceptuses

concerns the comparison of the 737 "exposed" conceptuses to the 2031 "unexposed".

Tables 3 and 4 provide crude and adjusted measures on Any Dioxin and TCDD Only. None of the odds ratios for Any Dioxin (Table 3) were significantly elevated, either crude or after adjustment for pertinant confounders. There was a notable lack of homogeneity among some of the subtables: all conceptus deaths, spontaneous abortions, and unfavorable outcomes.

For TCDD Only (Table 4), none of the odds ratios were significantly elevated. Notice that some of the odds ratios, All Conceptus Deaths and Infant Deaths, increased slightly after adjustment. Notice also that the subtables, for the same three dependent variables - All Conceptus Deaths, Spontaneous Abortions, and Unfavorable Outcomes, are marginally non-homogeneous.

The covariable-specific subtables for these exposure-outcome combinations that showed evidence of non-homogeneity were examined for significant associations, some 123 in number. Seven such associations were found; five with odds ratios greater than one, and two with odds ratios less than one. Recurrent, in the subtables with elevated odds ratios, was a group of 9 TCDD-exposed conceptuses who contributed 3 spontaneous abortions and only 6 live births to the analyses. Mothers of these conceptuses characteristically were 18-30 years of age, gravida 4+, were not using birth control methods

Table 3. Crude and Adjusted Odds Ratios, P Values and Homogeneity of Subtables, All Outcomes, Any Dioxin

| Outcome | Crude Or | Crude P | Adjusted Or | Adjusted P | Adjusted Homogenity (P) |
|---|---|---|---|---|---|
| All Conceptus Deaths | 1.14 | .31 | 1.03 | .44 | <.01 |
| Stillbirths | 1.27 | .26 | 1.06 | .49 | .20 |
| Spontaneous Abortions | 1.12 | .23 | 1.03 | .44 | .02 |
| Infant Deaths | 0.64 | .15 | 0.63 | .15 | .61 |
| Health Defects | 0.93 | .37 | 0.85 | .20 | .57 |
| Congenital Malformations | 0.96 | .48 | 0.85 | .62 | .62 |
| All Unfavorable Outcomes | 1.03 | .42 | 0.97 | .41 | .05 |

Table 4. Crude and Adjusted Odds Ratios, P Values, and Homogenity of Subtables, All Outcomes, TCDD Only

| Outcome | Crude Or | Crude P | Adjusted Or | Adjusted P | Adjusted Homogenity (P) |
|---|---|---|---|---|---|
| All Conceptus Deaths | 0.99 | .93 | 1.02 | .49 | .09 |
| Stillbirths | 1.03 | .54 | 0.97 | .56 | .37 |
| Spontaneous Abortions | 0.98 | .49 | 0.96 | .45 | .06 |
| Infant Deaths | 0.74 | .32 | 0.82 | .41 | .42 |
| Health Defects | 1.00 | .54 | 0.93 | .40 | .60 |
| Congenital Malformations | 1.18 | .30 | 1.08 | .43 | .86 |
| All Unfavorable Outcomes | 0.98 | .47 | 0.98 | .47 | .06 |

at conception, reported no illnesses during pregnancy, did not work at high risk jobs, took medications during pregnancy, and had fewer than seven drinks per week. Since TCDD Only was a subset of Any Dioxin, and Spontaneous Abortions was a subset of All Conceptus Deaths, which, in turn, was a subset of All Unfavorable Outcomes, it is probably not surprising that this one covariable-specific group produced such a ripple effect. I should note that a shift of even a single covariable eliminated statistical significance and, in some cases, changed the direction of the association.

Not shown are the results for the categories moderate-to-high TCDD and other dioxins above. Their numbers were quite small, but none of the results were statistically significant; each, of course, was a subset of at least one of the categories shown. Crude and adjusted trend analyses based on length-of-exposure categories were also non-significant.

## DISCUSSION

In summary, there were no statistically significant differences in crude or adjusted risk estimates for any of the pregnancy outcomes studied. Nor were there significant trands overall with increased duration of exposure.

The observed lack of association of adverse pregnancy outcomes with differences in paternal exposure to dioxins may have a variety of explanations. There may have been problems related to selection, misclassification, confounding or power that prevented us from picking up true differences in the two groups.[18,19] These are, of course, problems with any epidemiological study and are discussed in the referenced report.[16]

In addition, there may have been a problem with recall bias. That is, those among the wives of the exposed, having been informed of the intent of the study prior to the interview, may have ruminated about adverse outcomes and reported more, relatively, than did the controls. This would have produced an artificial elevation in some or all of the risk estimates.

It is also possible that the results reflect the true state of nature; that is, there is no risk to human reproduction caused by paternal exposures to dioxins. This explanation is consistent with most of the animal and human data we have to date.

In conclusion, it is our interpretation that, under the conditions of this study, there were no biologically meaningful associations between adverse pregnancy outcomes and paternal dioxin exposures.

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# A MORTALITY STUDY OF WORKERS EMPLOYED AT THE MONSANTO COMPANY PLANT IN NITRO, WEST VIRGINIA

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## BACKGROUND

The compound 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) is a highly toxic impurity that is formed in trace quantities during the production of 2,4,5-trichlorophenoxyacetic acid (2,4,5-T). Exposure to TCDD can cause chloracne, a skin disorder characterized by comedones, cysts, and abscesses.[1] Outbreaks of chloracne have been reported among workers associated with the production of 2,4,5-T and 2,4,5-T based products. Such incidents resulting from both accidental and routine occupational exposures have been reported from several countries.[2]

The first reported industrial accident involving exposure to TCDD occurred in 1949 at the Monsanto Company plant in Nitro, West Virginia. A total of 122 employees developed symptoms of chloracne following a trichlorophenol (TCP) process accident. An undetermined number of other employees developed symptoms of chloracne resulting from exposure to the regular operations concerned with 2,4,5-T production over the period 1948-1969.

Between 1949 and 1953, Ashe and Suskind[3,4] examined thirty-eight Nitro plant employees with chloracne. Twelve of these had developed symptoms of chloracne directly following the 1949 TCP accident; twenty-six other chloracne cases had resulted from exposure to the regular 2,4,5-T production operations. In addition to chloracne, other signs and symptoms were observed in this group. These included severe aches in the lower extremities, fatigue, nervousness and irritability, loss or decrease of libido, dyspnia, and vertigo. These findings are consistent with those that have been reported in other industrial incidents.[5]

To examine the chronic health effects of exposure to TCDD, a mortality study of the Nitro plant employees who had developed symptoms of chloracne following the 1949 TCP accident was conducted.[6] The study cohort was comprised of 121 of the 122 chloracne cases; one female who was living as of the endpoint of the study was excluded from analysis. At the time of the incident, it was assumed that the symptoms were caused by exposure to unknown products of decomposition. From today's vantage point, these symptoms suggest exposure to TCDD. The 121-member study cohort, with a presumptive high-peak exposure to TCDD, was followed for mortality through 1978. The entire cohort was traced: thirty-two deaths were observed and eighty-nine persons were confirmed as living. Analysis indicated no excess in total mortality or in deaths from malignant neoplasms.

The study presented here examines the mortality of Nitro plant workers who were assigned to an area of TCP or 2,4,5-T production, with potential for exposure to TCDD. The mortality of these workers is examined in the context of the mortality experience of the total Nitro plant worker population.

The Monsanto Nitro plant began operations in 1922, when the Rubber Service Laboratories purchased the plant as war surplus and began production of chemicals and additives for the growing rubber industry. In 1929, Monsanto Company purchased the Nitro plant from the Rubber Service Laboratories and entered the rubber chemicals business. Over the years, the Nitro plant has diversified to where it now produces agricultural chemicals, paper chemicals, plasticizers, fine chemicals, and intermediates, in addition to rubber chemicals. The plant is situated in the Kanawha River Valley, an area containing one of the largest concentrations of chemical production facilities in the United States.

Of the many chemicals used over the years at the Nitro plant, one has an established association with the occurrence of cancer in man. Para-aminobiphenyl (PAB), used from 1941 through 1952 for use as a rubber antioxidant and dye intermediate, was shown in 1954 by Walpole et al.[7] to induce bladder cancer in dogs. In 1955, Melick et al.[8] confirmed the carcinogenicity of para-aminobiphenyl to man with the reporting of bladder tumors among workers exposed to this chemical at two Monsanto plants. Para-aminobiphenyl was produced at one plant and then transferred by tank car to the Nitro plant where additional processing was carried out. The minimum duration of exposure reported to have produced a bladder tumor is 133 days; the latent period has ranged from 15 to 35 years.[9] An intensive screening program was instituted at Monsanto Company about 1955 to examine, on a continuing basis, all workers exposed to this chemical. Seven deaths from bladder cancer have occurred among Nitro plant employees enrolled in this program. These seven deaths are included in the present study.

Other chemicals with known health effects produced at the Nitro plant include methylparathion and carbon disulfide. The acute effects of exposure to each of these chemicals have been described[9,10] while the chronic effects are less well understood. Besides p-aminobiphenyl, several other rubber chemicals are of potential health concern. For all of these, there is insufficient evidence to evaluate their carcinogenicity to man. Tetramethyl thiuram disulfide is considered an animal carcinogen[11] and there is some evidence, although not sufficient, that N-methyl-N,4-dinitrosaniline is also an animal carcinogen.[12] For several other rubber chemicals, there is insufficient evidence to evaluate the carcinogenicity to animals. However, these chemicals have the potential for forming nitrosamines, certain of which are known animal carcinogens.[13] Zinc dimethyl dithiocarbamate, tetramethyl thiuram disulfide and tetramethyl thiuram monosulfide have the potential to form N-nitrosodimethylamine. N-Nitrosomorpholine has been found in product samples of 2-(morpholinothio) benzothiazole and 4,4'-dithiodimorpholine (Frisone, G.J., The General Tire and Rubber Company, unpublished data).

Although several of the chemical compounds produced or used at the Nitro plant over the years have been associated with adverse health effects, no attempt has been made to relate chemical exposure to mortality with the exception of decedents exposed to the TCP or 2,4,5-T operations and potentially exposed to TCDD. As a result, the only specific hypothesis that can be tested is whether a relationship exists between potential TCDD exposure and proportional mortality, especially for malignant neoplasms. The mortality for this group is examined in addition to that of the total Nitro plant worker population.

## POPULATION AND METHODS

A study cohort was developed from the Nitro plant consisting of employees active on or after January 1, 1955 with one or more years of employment on the hourly roll prior to December 31, 1977. Salaried personnel who had never worked on the hourly roll were excluded from study because many of these employees had no appreciable exposure to the plant environment and, for the most part, their exposure cannot be determined from plant records. Females and non-white males were also excluded because of their small numbers.

The cohort was assembled using government earnings reports, independent of the plant work history records. Annual earnings reports were available on a computer file from 1951 through 1977. Names and social security numbers were identified from this source. Work history records were used to supplement the earnings records. Information on race, sex, date of birth, date of hire, date of separation, and, if deceased, 2,4,5-T exposure was abstracted from these records. Ascertainment of 2,4,5-T exposure was confined to decedents only because it was too tedious to do for the entire cohort. The information from the work history records was used to determine which names identified from the annual earnings records met the cohort entrance criteria. Employees identified from the earnings records who terminated prior to 1955 were not included in this study because work history records for all such employees were not retained prior to this date.

Exposure to 2,4,5-T was determined by assignment to a 2,4,5-T operation based on the work history records. 2,4,5-T exposure was determined for all but one decedent. Employees holding a job having plant-wide responsibilities with the potential for exposure to 2,4,5-T were, for the purposes of this study, considered to be non-exposed.

The vital status of each member of the study cohort was determined using standard follow-up techniques and ascertained as of December 31, 1977. Death certificates were coded by an independent nosologist for the underlying cause of death, according to the rules of the Eighth Revision of the International Classification of Diseases, Adapted.[14]

Data for the total Nitro plant study population were analyzed by the modified life-table method using the U.S. population as the standard. With this method of analysis, the age-, race-, time-, and cause-specific mortality rates for the U.S. general population are applied to the person-years lived classified by age, race, and time. A standardized mortality ratio (SMR) was calculated for 23 selected cause of death categories. Cause-specific SMR's for 15 selected cancer sites were calculated for subgroups of the total Nitro plant study population defined by date of death, age at death, and year of hire. The statistical significance of the deviation in a SMR from 100 was tested using the formula:

$$\text{standard error of SMR} = \frac{100\sqrt{\text{no. observed deaths}}}{\text{no. expected deaths}}$$

If the observed SMR differed from 100 by 1.96 standard errors, it was regarded as significant at the 5% level. A SMR was tested for significance only when the observed number of deaths was five or greater.

Data for those deceased were also analyzed according to 2,4,5-T exposure using the proportional mortality method. In this case, the expected number of deaths is calculated on the basis of proportions of deaths observed in the U.S. general population. A proportional mortality ratio (PMR) was calculated for 23 selected cause-of-death categories. PMR's were calculated separately for those exposed to 2,4,5-T and for those not exposed. The statistical significance of the deviation of a PMR from 100 was tested by calculating the 95%

confidence interval for the PMR for a given cause k according to the formula:

$$\text{confidence interval for } PMR_k = \frac{N(\text{confidence limits for \% of observed deaths})}{exp_k}$$

where N = number of observed deaths for all causes

Both the standardized and proportional mortality analyses were conducted using the computer program developed by Monson.[15] The observed mortality was compared to that of the United States white male population for the time period of study.

RESULTS

A total of 884 men were identified for study and traced for deaths through 1977. The entire cohort was successfully traced. Death certificates were obtained for all deaths. Seven hundred twenty-one (82%) were verified as living and 163 (18%) were confirmed dead by death certificates.

Tables 1-3 characterize the total Nitro plant study population by age at hire, year of hire, and length of employment. The distribution of the study population by age at hire indicates that the workers were fairly young at first hire (Table 1). Seventy-two percent of the study cohort were hired by age 30. None were hired at age 50 or above.

Table 2 shows the distribution of the study population by year of hire. The majority of workers (75.3%) were hired during the period 1940-1959, while smaller percentages of workers were hired prior to 1940 (10.8%) and after 1960 (13.9%).

An examination of the study population by length of employment indicates a fairly even distribution over the intervals less than 10 years, 10-19 years, 20-29 years, and greater than 30 years (Table 3).

Observed and expected deaths occurring during 1955-1977 among the total Nitro plant study population are shown by cause in Table 4. The SMR for all causes of death was 103 with 163 deaths observed and 158.10 expected. There were 35 deaths from malignant neoplasms with 30.92 expected, yielding a SMR of 113. Significantly elevated SMR's were seen for the categories of malignant neoplasms of the genitourinary organs and of the bladder. The SMR for bladder cancer was 989 with 9 deaths observed and 0.91 expected. This excess in bladder cancer deaths is reflected in the elevated SMR for malignant nioplasms of the genitourinary organs. A significantly elevated SMR is also seen for arteriosclerotic heart disease. There were 79 deaths from this cause with 59.40 expected (SMR = 133). The SMR for

Table 1. Distribution of Total Nitro Plant Study Population by Age at Hire

| Age at Hire | Number | Percent |
|---|---|---|
| <20 | 122 | 13.8 |
| 20-29 | 518 | 58.6 |
| 30-39 | 191 | 21.6 |
| 40-49 | 53 | 6.0 |
| 50+ | 0 | 0.0 |
| Total | 884 | 100.0 |

Table 2. Distribution of Total Nitro Plant Study Population by Year of Hire

| Year of Hire | Number | Percent |
|---|---|---|
| Prior to 1930 | 21 | 2.4 |
| 1930-1939 | 74 | 8.4 |
| 1940-1949 | 361 | 40.8 |
| 1950-1959 | 305 | 34.5 |
| 1960-1976 | 123 | 13.9 |
| Total | 884 | 100.0 |

Table 3. Distribution of Total Nitro Plant Study Population by Length of Employment

| Length of Employment (yrs.) | Number | Percent |
|---|---|---|
| <10 | 225 | 25.5 |
| 10-19 | 213 | 24.1 |
| 20-29 | 204 | 23.1 |
| 30+ | 242 | 27.4 |
| Total | 884 | 100.1 |

Table 4. Observed and Expected Number of Deaths During 1955-1977 by Cause Showing Standardized Mortality Ratios (SMR'S) for Total Nitro Plant Study Population

| Cause of Death | ICDA Codes 8th Rev. | Observed | Expected | SMR |
|---|---|---|---|---|
| All causes of death | | 163 | 158.10 | 103 |
| All malignant neoplasms | 140-209 | 35 | 30.92 | 113 |
| Buccal cavity and pharynx | 140-149 | 0 | 1.03 | 0 |
| Digestive organs and peritoneum | 150-159 | 4 | 8.65 | 46 |
| Stomach | 151 | 1 | 1.63 | 61 |
| Liver | 155-156 | 0 | 0.60 | 0 |
| All other digestive organs | --- | 3 | 6.42 | 47 |
| Respiratory system | 160-163 | 14 | 10.51 | 133 |
| Lung | 162-163 | 14 | 9.91 | 141 |
| All other respiratory organs | --- | 0 | 0.60 | 0 |
| Skin | 172-173 | 0 | 0.58 | 0 |
| Genitourinary organs | 185-189 | 12 | 3.75 | 320* |
| Bladder | 188 | 9 | 0.91 | 989* |
| All other genitourinary organs | --- | 3 | 2.84 | 106 |
| Lymphatic and hematopoietic tissue | 200-209 | 1 | 3.15 | 32 |
| Other sites | --- | 4 | 3.25 | 123 |
| Diseases of the nervous system and sense organs | 320-389 | 0 | 1.28 | 0 |
| Diseases of the circulatory system | 390-458 | 92 | 82.59 | 111 |
| Arteriosclerotic heart disease, including CHD | 410-413 | 79 | 59.40 | 133* |
| All other diseases of the circulatory system | --- | 13 | 23.19 | 56* |
| Diseases of the respiratory system | 460-519 | 6 | 9.13 | 66 |
| Diseases of the digestive system | 520-577 | 5 | 8.03 | 62 |
| All other diseases | --- | 5 | 10.46 | 48* |
| External causes of death | 800-998 | 20 | 15.69 | 127 |

Number at risk: 884

Person-years at risk: 13968.7

*p < .05

other circulatory diseases was significantly low at 56. A significant deficit was also seen for the category of all other diseases where the SMR was 48 with 5 deaths observed and 10.46 expected.

Trends for malignant neoplasm deaths with calendar time are shown in Table 5. SMR's which increased consistently over the period 1955-1977 are seen for the categories of all malignant neoplasms and malignant neoplasms of the respiratory system. The SMR for all malignant neoplasms rose from 32 to 131. The SMR for malignant neoplasms of the respiratory system rose from 0 to 172 reflecting a SMR for lung cancer which increased from 0 to 181. Although based on very small numbers, the SMR for malignant neoplasms of the digestive organs and peritoneum consistently decreased over time from 99 to 24. The SMR for malignant neoplasms of the genitourinary organs peaked during the period 1960-1969. This is reflected in the SMR for bladder cancer which increased from 0 in 1955-1959 to a peak of 1471 in 1960-1969 and decreased to 833 in 1970-1977.

The analysis of observed and expected deaths from malignant neoplasms by age at death revealed little in terms of consistent trends with age (Table 6). For most of the cause-of-death categories, the SMR peaked at age 45-64 rather than continuing to rise with increasing age. Bladder cancer is one category where the SMR remained high at age 65 and over.

The distribution of deaths from malignant neoplasms by year of hire is shown in Table 7. No deaths from malignant neoplasms were observed among workers hired after 1960. The SMR for all malignant neoplasms was similar for those hired prior to 1945 and for those hired in the period 1945-1959. The greatest difference in SMR's between those hired prior to 1945 and those hired from 1945-1959 occurs with bladder cancer. The SMR for bladder cancer is highest for those hired prior to 1945 with a SMR of 1111.

A subset of deaths identified from the total Nitro plant study population was studied separately. Table 8 characterizes the decedents according to 2,4,5-T exposure. Of the 163 decedents, 58 (35.6%) were considered to be exposed to 2,4,5-T based on their work history records, while 104 (63.8%) were considered to be non-exposed. The exposure of one decedent was unkown.

The results of the proportional mortality analysis by 2,4,5-T exposure classification are presented in Table 9. The proportion of cancer deaths among 2,4,5-T workers is lower than in the non-exposed group (PMR: 82 vs. 122). The PMR for lung cancer deaths is slightly higher in the exposed group (PMR: 159 vs. 117). The proportion of deaths due to bladder cancer is higher among the decedents not exposed to 2,4,5-T. There were 7 deaths from bladder cancer among these workers with 0.65 expected (PMR = 1077). The PMR for bladder cancer among decedents exposed to 2,4,5-T was 909 with 2 deaths observed and

Table 5. Observed and Expected Deaths from Malignant Neoplasms During 1955-1977 by Calendar Time Showing Standardized Mortality Ratios (SMR'S) for Total Nitro Plant Study Population

| | Calendar Time | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | 1955-1959 | | | 1960-1969 | | | 1970-1977 | | |
| Cause of Death | Observed | Expected | SMR | Observed | Expected | SMR | Observed | Expected | SMR |
| All malignant neoplasms | 1 | 3.13 | 32 | 13 | 11.81 | 110 | 21 | 15.98 | 131 |
| Buccal cavity and pharynx | 0 | 0.11 | 0 | 0 | 0.41 | 0 | 0 | 0.50 | 0 |
| Digestive organs and peritoneum | 1 | 1.01 | 99 | 2 | 3.48 | 57 | 1 | 4.16 | 24 |
| Stomach | 0 | 0.25 | 0 | 0 | 0.70 | 0 | 1 | 0.69 | 145 |
| Liver | 0 | 0.09 | 0 | 0 | 0.28 | 0 | 0 | 0.23 | 0 |
| All other digestive organs | 1 | 0.67 | 149 | 2 | 2.50 | 80 | 0 | 3.24 | 0 |
| Respiratory system | 0 | 0.87 | 0 | 4 | 3.83 | 104 | 10 | 5.81 | 172 |
| Lung | 0 | 0.80 | 0 | 4 | 3.59 | 111 | 10 | 5.52 | 181 |
| All other respiratory organs | 0 | 0.07 | 0 | 0 | 0.24 | 0 | 0 | 0.29 | 0 |
| Skin | 0 | 0.07 | 0 | 0 | 0.23 | 0 | 0 | 0.28 | 0 |
| Genitourinary organs | 0 | 0.33 | 0 | 5 | 1.37 | 365 | 7 | 2.05 | 341 |
| Bladder | 0 | 0.09 | 0 | 5 | 0.34 | 1471* | 4 | 0.48 | 833 |
| All other genitourinary organs | 0 | 0.24 | 0 | 0 | 1.03 | 0 | 3 | 1.57 | 191 |
| Lymphatic and hematopoietic tissue | 0 | 0.40 | 0 | 0 | 1.24 | 0 | 1 | 1.50 | 67 |
| Other sites | 0 | 0.34 | 0 | 2 | 1.25 | 160 | 2 | 1.68 | 119 |

*$p < .05$

Table 6. Observed and Expected Deaths from Malignant Neoplasms During 1955-1977 by Age at Death Showing Standardized Mortality Ratios (SMR'S) for Total Nitro Plant Study Population

| | Age at Death | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | <45 | | | 45-64 | | | 65+ | | |
| Cause of Death | Observed | Expected | SMR | Observed | Expected | SMR | Observed | Expected | SMR |
| All malignant neoplasms | 2 | 2.39 | 84 | 21 | 16.85 | 125 | 12 | 11.69 | 103 |
| Buccal cavity and pharynx | 0 | 0.06 | 0 | 0 | 0.67 | 0 | 0 | 0.31 | 0 |
| Digestive organs and peritoneum | 0 | 0.51 | 0 | 4 | 4.58 | 87 | 0 | 3.57 | 0 |
| Stomach | 0 | 0.10 | 0 | 1 | 0.85 | 118 | 0 | 0.68 | 0 |
| Liver | 0 | 0.03 | 0 | 0 | 0.32 | 0 | 0 | 0.24 | 0 |
| All other digestive organs | 0 | 0.38 | 0 | 3 | 3.41 | 88 | 0 | 2.65 | 0 |
| Respiratory system | 0 | 0.54 | 0 | 11 | 6.36 | 173 | 3 | 3.61 | 83 |
| Lung | 0 | 0.51 | 0 | 11 | 5.99 | 184 | 3 | 3.42 | 88 |
| All other respiratory organs | 0 | 0.03 | 0 | 0 | 0.37 | 0 | 0 | 0.19 | 0 |
| Skin | 0 | 0.13 | 0 | 0 | 0.31 | 0 | 0 | 0.14 | 0 |
| Genitourinary organs | 1 | 0.20 | 500 | 3 | 1.43 | 210 | 8 | 2.12 | 377* |
| Bladder | 1 | 0.02 | 5000 | 2 | 0.39 | 513 | 6 | 0.50 | 1200* |
| All other genitourinary organs | 0 | 0.18 | 0 | 1 | 1.04 | 96 | 2 | 1.62 | 123 |
| Lymphatic and hematopoietic tissue | 0 | 0.55 | 0 | 1 | 1.60 | 63 | 0 | 1.00 | 0 |
| Other sites | 1 | 0.40 | 250 | 2 | 1.90 | 105 | 1 | 0.94 | 106 |

*p < .05

Table 7. Observed and Expected Deaths from Malignant Neoplasms During 1955-1977 by Year of Hire Showing Standardized Mortality Ratios (SMR'S) for Total Nitro Plant Study Population

| Cause of Death | Observed | Expected | SMR | Observed | Expected | SMR | Observed | Expected | SMR |
|---|---|---|---|---|---|---|---|---|---|
| All malignant neoplasms | 25 | 21.30 | 117 | 10 | 8.63 | 116 | 0 | 0.99 | 0 |
| Buccal cavity and pharynx | 0 | 0.70 | 0 | 0 | 0.31 | 0 | 0 | 0.03 | 0 |
| Digestive organs and peritoneum | 2 | 6.27 | 32 | 2 | 2.19 | 91 | 0 | 0.19 | 0 |
| Stomach | 0 | 1.21 | 0 | 1 | 0.39 | 256 | 0 | 0.03 | 0 |
| Liver | 0 | 0.45 | 0 | 0 | 0.14 | 0 | 0 | 0.01 | 0 |
| All other digestive organs | 2 | 4.61 | 43 | 1 | 1.66 | 60 | 0 | 0.15 | 0 |
| Respiratory system | 10 | 7.14 | 140 | 4 | 3.09 | 129 | 0 | 0.27 | 0 |
| Lung | 10 | 6.73 | 149 | 4 | 2.93 | 137 | 0 | 0.26 | 0 |
| All other respiratory organs | 0 | 0.41 | 0 | 0 | 0.16 | 0 | 0 | 0.01 | 0 |
| Skin | 0 | 0.31 | 0 | 0 | 0.22 | 0 | 0 | 0.05 | 0 |
| Genitourinary organs | 9 | 2.90 | 310* | 3 | 0.76 | 395 | 0 | 0.09 | 0 |
| Bladder | 8 | 0.72 | 1111* | 1 | 0.18 | 556 | 0 | 0.01 | 0 |
| All other genitourinary organs | 1 | 2.18 | 46 | 2 | 0.58 | 345 | 0 | 0.08 | 0 |
| Lymphatic and hematopoietic tissue | 1 | 1.95 | 51 | 0 | 1.00 | 0 | 0 | 0.20 | 0 |
| Other sites | 3 | 2.03 | 148 | 1 | 1.06 | 94 | 0 | 0.16 | 0 |

*p < .05

Table 8. 2,4,5-T Exposure Classification for Decendents During 1955-1977 Among Total Nitro Plant Study Population

| 2,4,5-T Exposure Classification | Number of Deaths | Percent |
|---|---|---|
| Exposed | 58 | 35.6 |
| Non-exposed | 104 | 63.8 |
| Unknown | 1 | 0.6 |
| Total | 163 | 100.0 |

Table 9. Observed and Expected Number of Deaths During 1955-1977 by Cause and 2,4,5-T Exposure Category Showing Proportional Mortality Ratios (PMR'S)

| | 2,4,5-T Exposure Category | | | | | |
|---|---|---|---|---|---|---|
| | Exposed | | | Non-exposed | | |
| Cause of Death | Observed | Expected | PMR | Observed | Expected | PMR |
| All malignant neoplasms | 9 | 10.94 | 82 | 25 | 20.43 | 122 |
| Buccal cavity and pharynx | 0 | 0.38 | 0 | 0 | 0.64 | 0 |
| Digestive organs and peritoneum | 0 | 2.80 | 0 | 3 | 5.74 | 52 |
| Stomach | 0 | 0.52 | 0 | 0 | 1.06 | 0 |
| Liver | 0 | 0.19 | 0 | 0 | 0.39 | 0 |
| All other digestive organs | 0 | 2.09 | 0 | 3 | 4.29 | 70 |
| Respiratory system | 6 | 3.78 | 159 | 8 | 6.81 | 117 |
| Lung | 6 | 3.57 | 168 | 8 | 6.42 | 125 |
| All other respiratory organs | 0 | 0.21 | 0 | 0 | 0.39 | 0 |
| Skin | 0 | 0.29 | 0 | 0 | 0.35 | 0 |
| Genitourinary organs | 2 | 0.96 | 208 | 10 | 2.70 | 370* |
| Bladder | 2 | 0.22 | 909 | 7 | 0.65 | 1077* |
| All other genitourinary organs | 0 | 0.74 | 0 | 3 | 2.05 | 146 |
| Lymphatic and hematopoietic tissue | 0 | 1.35 | 0 | 1 | 2.07 | 48 |
| Other sites | 1 | 1.38 | 72 | 3 | 2.12 | 142 |
| Diseases of the nervous system and sense organs | 0 | 0.61 | 0 | 0 | 0.83 | 0 |
| Diseases of the circulatory system | 31 | 26.48 | 117 | 61 | 55.34 | 110 |
| Arteriosclerotic heart disease, including CHD | 27 | 19.72 | 137 | 52 | 39.68 | 131* |
| All other diseases of the circulatory system | 4 | 6.76 | 59 | 9 | 15.66 | 57 |
| Diseases of the respiratory system | 2 | 2.67 | 75 | 4 | 6.49 | 62 |
| Diseases of the digestive system | 1 | 3.70 | 27 | 4 | 4.93 | 81 |
| All other diseases | 3 | 4.31 | 70 | 2 | 6.70 | 30 |
| External causes of death | 12 | 9.29 | 129 | 8 | 9.28 | 86 |
| Total number of deaths: | 58 | 58.00 | | 104 | 104.00 | |

*p < .05

0.22 expected. PMR's for both exposure groups are quite similar for diseases of circulatory and respiratory system. Slight differences appear in the PMR's between the two groups for diseases of the digestive system and all other diseases. However, the PMR's for both groups are quite low. The PMR for external causes of death is slightly higher in the exposed group (PMR: 129 vs. 86).

A listing of the cancer deaths among the 2,4,5-T exposed is given in Table 10. Table 11 listed the cancer deaths among the non-exposed group.

## DISCUSSION

The observation made many years ago of an apparent excess in bladder cancer among Nitro plant workers was confirmed and quantified in the mortality analysis of the total Nitro plant population presented here. The SMR for bladder cancer was 989 and was the only statistically significant SMR among those for malignant neoplasms. The excess in mortality is not seen until 1960. The SMR peaked in the 1960's and declined somewhat in the 1970's. The excess also appeared to be clustered in those decedents aged 65 years and older at death and in those hired prior to 1945. This would suggest that we should see a further decline in the SMR for bladder cancer over time.

Although not statistically significant, the SMR for lung cancer appears to be elevated. The SMR increased with calendar time and was highest in those hired prior to 1945. The elevation appears to be clustered in those aged 45-64 years of age at death (SMR = 184) and does not show a gradient with age. Further analyses to evaluate trends in lung cancer deaths as they relate to occupation cannot be carried out due to limitations in the data collected in this study.

The SMR for diseases of the circulatory system was elevated at 111. This is most likely a reflection of the higher mortality from heart disease which has been observed for Charleston, West Virginia and Kanawha County, West Virginia (unpublished data, Neas LM, 1979 and Enterline PE, 1979). For the total Nitro plant study population, there was a statistically significant excess in deaths from arteriosclerotic heart disease and a deficit in deaths from other circulatory diseases. This variation in the distribution of deaths from that of the U.S. may be due to risk factor or medical care differences in the plant population and the local area. These may include differences in smoking habits, the availability and use of medical services and the specificity of diagnoses.

The proportional mortality analysis of decedents by 2,4,5-T exposure classification indicated no unusual patterns of mortality in the 2,4,5-T exposed. The proportional mortality ratio (PMR for malignant neoplasms was low (PMR = 82) in the exposed group. Lung cancer was the only site among the malignant neoplasms which was somewhat higher in the exposed group.

Table 10. Deaths Due to Malignant Neoplasms Among Nitro Plant Workers Exposed to 2,4,5-T

| Year of Birth | Year of Hire | Year of 1st Exposure | Year of Term. | Year of Death | Smoking History* | Cause of Death as Given on Death Certificate |
|---|---|---|---|---|---|---|
| 1917 | 1946 | 1951 | 1972 | 1972 | Cigarettes | Carcinoma left lung with metastases (162.1) |
| 1911 | 1948 | 1955 | 1972 | 1972 | Cigarettes | Metastatic carcinoma of the lung (162.1) |
| 1916 | 1946 | 1959 | 1968 | 1968 | Cigarettes | Bronchiogenic carcinoma of right upper lobe (162.1) |
| 1901 | 1944 | 1956 | 1963 | 1973 | Cigarettes | Carcinoma lung with metastases (162.1) |
| 1911 | 1941 | 1948 | 1971 | 1975 | Cigarettes | Bronchiogenic carcinoma with cerebral metastases (162.1) |
| 1922 | 1945 | 1948 | 1972 | 1973 | Non-smoker | Bronchiogenic carcinoma with metastases (162.1) |
| 1923 | 1946 | 1950 | 1972 | 1972 | Cigarettes | Generalized liposarcoma (171.9) |
| 1902 | 1922 | 1948 | 1966 | 1966 | Non-smoker | Metastatic carcinoma urinary bladder (188.0)** |
| 1910 | 1944 | 1948 | 1968 | 1968 | Non-smoker | Carcinoma of the urinary bladder (188.0)** |

* Obtained by interview with former coworkers of decedents.
** Included on the Nitro plant PAB roster.

Table 11. Deaths Due to Malignant Neoplasms Among Nitro Plant Workers Not Exposed to 2,4,5-T

| Year of Birth | Year of Hire | Year of Termin. | Year of Death | Smoking History* | Cause of Death as Given on Death Certificate |
|---|---|---|---|---|---|
| 1911 | 1945 | 1966 | 1968 | Cigarettes | Carcinoma of colon (153.8) |
| 1904 | 1935 | 1957 | 1957 | Cigarettes | Carcinoma of liver (157.9) |
| 1899 | 1929 | 1959 | 1960 | Pipe | Intraperitoneal carcinoma (158.9) |
| 1905 | 1944 | 1970 | 1972 | Cigarettes | Carcinoma of lung (162.1) |
| 1909 | 1943 | 1962 | 1962 | Cigarettes | Carcinoma of left lung (162.1) |
| 1910 | 1927 | 1966 | 1970 | Cigarettes | Pulmonary carcinoma (162.1) |
| 1912 | 1944 | 1965 | 1965 | Cigarettes | Carcinoma of apex of right lung (162.1) |
| 1915 | 1937 | 1977 | 1977 | Cigarettes | Carcinoma of lung (162.1) |
| 1915 | 1939 | 1969 | 1970 | Cigarettes | Carcinoma of lungs (162.1) |
| 1894 | 1944 | 1960 | 1964 | Non-smoker | Carcinoma of lung (162.1) |
| 1912 | 1937 | 1973 | 1974 | Cigarettes | Carcinoma of lung (162.1) |
| 1919 | 1946 | 1963 | 1964 | Cigarettes | Carcinoma of urinary bladder (188.0)** |
| 1901 | 1933 | 1962 | 1977 | Cigarettes | Carcinoma of bladder (188.0)** |
| 1897 | 1941 | 1962 | 1965 | Cigarettes | Carcinoma of urinary bladder (188.0)** |
| 1898 | 1933 | 1962 | 1965 | Smoked years ago | Carcinoma of urinary bladder (188.0)** |
| 1905 | 1943 | 1971 | 1975 | Cigarettes | Carcinoma of bladder (188.0) |
| 1898 | 1943 | 1963 | 1970 | Cigarettes | Bladder tumor (188.0)** |
| 1888 | 1944 | 1956 | 1970 | Unknown | Carcinoma of bladder (188.0) |
| 1905 | 1933 | 1969 | 1977 | Cigars | Prostatic carcinoma (185.0) |
| 1906 | 1946 | 1968 | 1977 | Cigarettes | Carcinoma of prostate (185.0) |
| 1925 | 1945 | 1973 | 1974 | Smoked years ago | Carcinoma of prostate (185.0) |
| 1919 | 1943 | 1972 | 1973 | Cigarettes | Hodgkin's Disease (201.0) |
| 1901 | 1941 | 1964 | 1965 | Cigars | Osteosarcoma arising from left arm (170.4) |
| 1901 | 1943 | 1966 | 1977 | Cigarettes | Carcinoma of liver & pancreas (197.8) |
| 1922 | 1944 | 1964 | 1964 | Cigarettes | Adenocarcinoma (199.0) |

* Obtained by interview with former coworkers of decedents.

** Included on the Nitro plant PAB roster.

The PMR analysis is limited in that an assessment of the total force of mortality cannot be made. The cause-specific PMR's only approximate what an SMR analysis would have produced.[16] The PMR analysis presented here estimates the cause-specific risks associated with 2,4,5-T exposure and potential TCDD exposure.

It is interesting to compare the results of this study of Nitro plant workers potentially exposed to TCDD with the results of the study of Nitro workers involved in the 1949 TCP accident. The workers involved in that incident had presumed TCDD exposure as evidenced by chloracne. The results of the two studies are similar in that neither shows an excess in deaths from any site among malignant neoplasms.

A recent study of Ott et al.[17] found no excess in total mortality or in deaths from malignant neoplasms among workers exposed to 2,3,5-T. These workers were probably exposed to very low levels of TCDD since no cases of chloracne were observed. Other studies of workers who developed chloracne resulting from TCDD exposure have been conducted and have been reviewed.[6] At the present time, data from these various studies do not constitute corroborative evidence of a cancer risk to man for any particular cancer site.

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# THE INFLUENCE OF PICLORAM (4-AMINO-3,5,6-TRICHLOROPICOLINIC ACID) ON SOME DIURNAL ORGANIC CONSTITUENT VARIATIONS IN CONVOLVULUS ARVENSIS AND KOCHIA SCOPARIA

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A DISSERTATION

submitted in partial fulfillment of the

requirements for the degree

DOCTOR OF PHILOSOPHY

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Circular 10-83-38

March 1, 1983

**TO:** Regional Directors; Directors, VA Medical Center Activities, Domiciliary, Outpatient Clinics, and Regional Offices with Outpatient Clinics (136)

**SUBJ:** Possible Exposure of Veterans to Herbicides During the Vietnam War, RCS 11-49

1. This represents a revision of Circular 10-81-54, dated March 19, 1981. The following circular is referenced: 10-82-37 dated March 15, 1982.

2. The issue of Agent Orange continues to be a genuine concern to a large number of veterans, the scientific community and the chemical industry, and as a result, continues to receive extensive media attention. The VA remains in the forefront of this issue and continues to play a leading role in supporting scientific and educational initiatives in an effort to provide all concerned veterans with the information and guidance they need, as well as any medical care for which they are eligible.

3. The Agent Orange Projects Office (10A7) has the responsibility to coordinate and monitor all DM&S activities relating to the Agent Orange issue including the registry. All policy and clinical questions relating to the potential effects of herbicides should be referred to this office (FTS: 389-5412). Questions relating to eligibility of veterans or treatment of active duty personnel should be referred to Medical Administration Service (136) VACO (FTS: 389-2598/2849).

4. The maintenance of the Agent Orange Registry remains an important function of the VA and is managed centrally by the Agent Orange Projects Office (10A7). The Agent Orange Registry remains our most effective means of identifying concerned Vietnam veterans. The importance of the role of each VA employee, beginning with the initial contact, in providing physical examinations and necessary treatment and advising the veteran of the results of the examination cannot be over-stressed. Any eligible Vietnam veteran expressing a concern relating to exposure to herbicides is encouraged to participate in the registry which includes a thorough medical examination. In addition, any eligible Vietnam veteran currently receiving treatment in VA medical centers and outpatient clinics will be identified and provided with the opportunity to participate in the Agent Orange Registry. Follow-up of the veterans entered into the registry will be conducted over a period of years in an effort to obtain further information regarding any long-term health effects resulting from these chemicals.

THIS CIRCULAR EXPIRES ON FEBRUARY 29, 1984

5. VA Environmental Physicians play a most significant role in determining the perceptions Vietnam veterans have concerning the quality of VA health care services and of their individual treatment by VA health care providers. The Environmental Physician will review the records of every Vietnam veteran examined to assure that a complete physical examination was performed and documented. It is important that each veteran be fully advised of the limitations of an Agent Orange related examination, that is, what the examination can or cannot reveal as regards the presence of dioxin in the body system and/or the relationship to adverse health effects or potential health defects or illnesses which may or may not be related to a veterans exposure to Agent Orange. I wish to strongly encourage your consideration of the best way to accomplish this communication process. The following alternatives might be considered.

(1) Provide each Vietnam veteran reporting to the Outpatient Admissions area with a handout describing the purpose of the examination and its limitations. This can be further clarified by the examining physician during the course of the physical examination, preferably prior to beginning the physical examination process.

(2) Provide each veteran with the opportunity to view the audiovisual "Agent Orange: A Search for Answers." Veterans and/or visitors to VA health care facilities should be informed concerning the film and when and where it can be viewed.

(3) Make all Agent Orange pamphlets and other informational materials available to Vietnam veterans and the public - keeping them displayed in prominent areas and ensuring that sufficient copies are available for distribution. It should be standard operating procedure to provide copies of VA Agent Orange pamphlets to all telephone callers requesting Agent Orange information.

6. It is essential that a complete medical history and physical examination be performed and documented. The medical history should be documented on SF 50 4 and SF 505 and the physical examination should be documented on SF 506 or VAF 10-7978e. The Agent Orange Registry code sheet (VAF 10-9009) does not replace any medical record. In eliciting the medical history and performing the physical examination (which should be conducted by/or under the direct supervision of the Environmental Physician), special attention will be given to those organ systems alleged to be most frequently

affected by exposure to herbicides containing TCDD. These include the liver, kidneys, skin and the reproductive, endocrine, immunologic and nervous systems. Particular attention will be paid to the detection of chloracne, a skin condition which has been associated with acute exposure to TCDD and other dioxins. Evidence will also be sought concerning the following potentially relevant symptoms or conditions: altered sex drive; sterility; congenital deformities among children; repeated infections; neoplasia; and for female veterans, difficulties in carrying pregnancies to term. In gathering these data, it is important to determine and record the time of onset of the symptoms or conditions; their intensity; the degree of physical incapacitation; and the details of any treatment received. The person actually performing the physical exam should be identified with the signature and title (M.D., P.A., etc.). If the examiner is other than a physician, a physician's countersignature is required, preferably the Environmental Physician. When an Agent Orange examination is done as part of a compensation and pension examination, the physical examination will be done by/or under the direct supervision of the Environmental Physician.

7. All veterans participating in the Agent Orange Registry will be given the following baseline laboratory studies: complete blood count, urinalysis, SMA-6, SMA-12, and a chest x-ray if one has not been done within the past 6 months. Appropriate additional diagnostic studies should be performed and consultations obtained as indicated by the patient's symptoms and physical and laboratory findings. Non-routine diagnostic studies, such as sperm counts, should be performed only if medically indicated.

8. The Environmental Physician will personally discuss with each veteran examined the results of the examination and the laboratory studies which are available at the time the physical examination is completed. This personal interview will be conducted in such a way as to encourage the veteran to discuss his/her own health concerns as well as those of his /her family as they relate to exposure to herbicides. In the absence of the Environmental Physician, the interview will be provided by a designated physician familiar with the Agent Orange program. The interviewing physician will document this action in a progress note in the veteran's medical record. In addition to the personal interview, a follow-up letter will be sent to each veteran explaining the results of the examination and laboratory studies. A copy of this letter will be filed in the veteran's administrative medical record. Recommended sample letters are provided in attachments A and B.

9. Particular attention is directed to the Special Registry at the Armed Forces Institute of Pathology (see DM&S Circular, dated 10-82-37 March 15, 1982). All pathological material (autopsy, surgical, cytologic, or other similar tissue) obtained from any Vietnam veteran will be processed in accordance with DM&S Circular 10-82-37 for inclusion in this special registry.

10. It has been determined that the analytical technology for measuring minute levels (parts per trillion) of TCDD in human fat does exist. The results of this study, however, are inconclusive as regards exposure to herbicides in Vietnam. Therefore, no VA medical center will perform any surgical or other procedure for the purpose of obtaining tissue for measuring TCDD in patients without prior approval by VACO (10A7).

11. When a Vietnam veteran requests an Agent Orange examination at a VA medical center, the center's Medical Administration Service will be notified and will initiate the procedures listed below:

a. Prepare a 3x5 card with the following typewritten information:

(1) Veteran's full name
(2) Veteran's address and telephone number
(3) Date of birth
(4) Social Security Number
(5) Date of initial examination
(6) Dates of follow-up examinations

b. The card will be filed alphabetically in a special file, labeled "Agent Orange Registry." This registry card will be maintained until further notice. Every effort should be made to maintain the veteran's current address and telephone number.

c. VA Form 10-10M contains a statement regarding "Possible Exposure." This item should be completed for all veterans applying for the Agent Orange examination.

12. The original records of all examinations performed on Vietnam veterans for possible herbicide toxicity are to be retained in the veteran's Consolidated Health Record (CHR). If a CHR does not already exist for a veteran examined for herbicide toxicity, one will be established, and the results of the examination for herbicide toxicity is to be enclosed in the CHR. A locator card will be created with the establishment of CHR.

13. The following procedures pertain to active duty personnel according to the site of the Agent Orange examination:

a. When active duty members of the uniformed services apply to VA facilities for an Agent Orange examination, the requirements of M-1, Part 1, Chapter 15 regarding the authorization from the appropriate branch of service and the billing of the appropriate branch of service will apply. The procedures of establishing a 3x5 card, of processing and completing the code sheet for active duty personnel will be the same as those followed for a veteran participating in the Agent Orange registry.

b. However, a military facility may perform the Agent Orange examination according to VA instructions. Military facilities have been informed to obtain a copy of the pertinent VA directive and samples of appropriate forms from the nearest VA facility. The completed physical examination, laboratory tests, and questionnaire will be forwarded to the nearest VA medical center or outpatient clinic. For these individuals the Medical Administration Service personnel at the medical center will:

(1) Prepare a colored 3x5 card with similar data as prepared for a veteran clearly label card as "Active Duty." Insert card in Agent Orange Registry file.
(2) Abstract the data from the medical record documents to the code sheet.
(3) Submit original code sheet to the VA Data Processing Center, Austin, TX as indicated in paragraph 18.
(4) Forward copies of the medical record documents with a copy of the code sheet to VA Central Office (10A7A).
(5) Place the original medical record documents in a plain folder properly identified with the name and social security number and a notation "Active Duty - Agent Orange Exam at ____________ military facility." These folders should be maintained in a special location in the file room.
(6) While the medical documents are not placed in the CHR (Type I or II folders), these special folders are subject to the same retention and disposition policies of the CHR.
(7) If an active-duty military [illegible] becomes discharged and reports for treatment [illegible] the Agent Orange examination will be filed [illegible]

14. There is a high priority concern for [illegible] handling and scheduling Agent Orange examinations. [illegible] should make every effort not to have 50 or more Agent [illegible] examinations pending at the end of the month. Each [illegible] 0 or more examinations pending will be contacted [illegible] Orange Projects Office Staff to ascertain the plan [illegible] be implemented in reducing the backlog and to determine how many examinations are pending beyond 30 days.

5

15. A monthly submission of medical record documents and code sheets will be sent to VACO, Agent Orange Projects Office (10A7A, Rm B-67) according to the mailing schedule listed in paragraph 17. The monthly submission will contain the following:

a. One legible copy of all the medical record documents relating to the Agent Orange examination. These documents should be placed in alphabetical order with the code sheets stapled on top of the medical record documentation for submission to 10A7A. Pertinent laboratory data and consultations obtained as part of these examinations will be held pending arrival of these data. Only copies of completed examinations should be submitted.

b. Follow-up examinations will be reported in the same manner.

c. The Agent Orange Registry Code Sheet (VAF 10-9009) will be prepared in three copies. One copy will be filed in the veteran's CHR with the documentation from the Agent Orange examination. One copy will be stapled to the corresponding medical record documents that are sent to VACO (10A7A). The original code sheet will be sent to the Austin Data Processing Center in (DPC). See paragraph 18 for instructions for mailing the code sheets to the DPC.

16. Instructions for completing the code sheets (VAF 10-9009) are listed in Attachment C. Effective with the issuance of this circular, the new code sheets (VAF 10-9009) must now be used. The Agent Orange Registry code sheet has been revised. DO NOT USE the VAF 10-9009 with November 1980 and September 1981 dates, they will no longer be accepted. The VAF 10-9009 with the 1982 date will be used. Old stocks of 1980 and 1981 VAF 10-9009 may be destroyed. The VAF 10-20681 (NR) Initial Data Base will no longer be used.

17. The following mailing schedule should be used for mailing the monthly submission to VACO (10A7A) and the Austin DPC.

| Region Number | Mailing Date |
|---|---|
| 1 | 6th of Month |
| 2 | 10th of Month |
| 3 | 14th of Month |
| 4 | 18th of Month |
| 5 | 22nd of Month |
| 6 | 26th of Month |

18. The following instructions should be followed for the mailing of the original code sheet (VAF 10-9009) to the DPC. These code sheets will be mailed monthly. Code sheets must be received at the DPC according to the mailing schedule listed in paragraph 17.

a. Batching of input documents:

(1) Code sheets should be scanned to ensure all required fields have been completed.

(2) Completed code sheets will be batched in groups of no more than 25 code sheets. Each batch will include code sheets for only one facility as indicated by identical entries in all six positions of the code sheet field one. Different facilities must be batched separately (i.e. VAMC one batch, OPC one batch). Batches of less than 25 code sheets are acceptable.

(3) Attach a transmittal form to each batch of documents. Record the six position facility number and the number of documents on the transmittal.

(4) Using the batch control log (see 18c below) assign the next sequential batch number and record it on the transmittal form. NOTE: Begin batching with batch number 001 in January of each year and continue with sequential numbers throughout the year.

(5) Code sheets should be stapled together in the upper left-hand corner. No medical record documentation should be attached to these code sheets.

(6) Corrected code sheets do not have to be batched separately or handled separately. They can be mailed with the regular code sheets as long as they are for the same facility number.

b. Transmittal form:

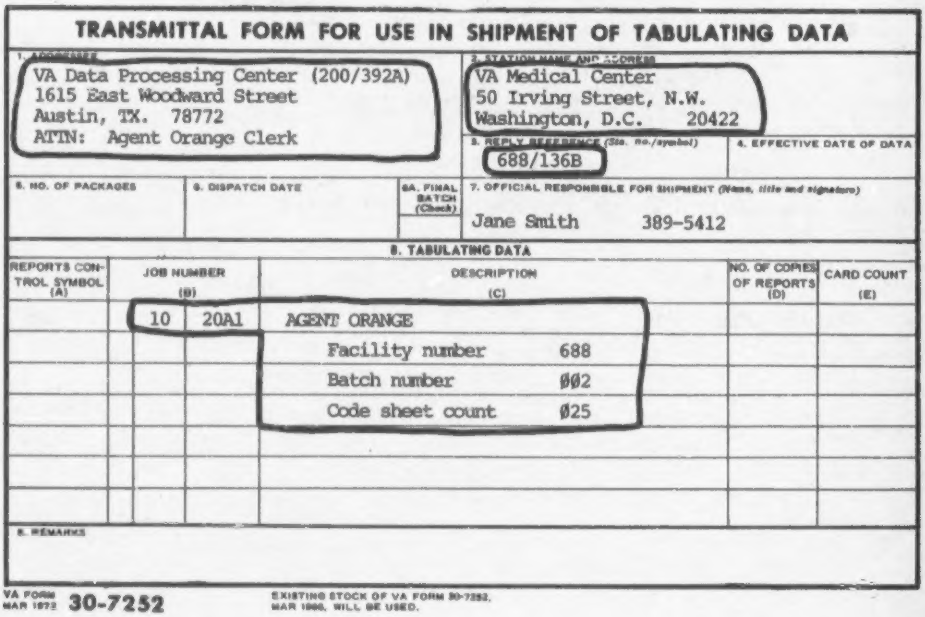
(1) Two copies of VA Form 30-7252, "Transmittal Form for the Use in Shipment of Tabulating Data," will accompany each batch of code sheets. One copy will be retained at

the Austin DPC and the other copy will be returned to the transmitting facility with the code sheets and edit analysis lists prepared at the DPC.

(2) The transmittal form will be completed as follows:

| | |
|---|---|
| Item 2: | Name and address of transmitting facility |
| Item 3: | Facility number of transmitting facility and correspondence symbol |
| Item 6: | Date of dispatch |
| Item 7: | Name and telephone number (FTS) of responsible individual at facility |
| Item 8: | Facility Number: The three (3) to six (6) position PTF facility number used on the code sheets of this batch (code sheet field #1)<br>Batch number: The batch number assigned by the transmitting facility (see below—control log).<br>Code Sheet Count: The number of code sheets in this batch (25 or less). |

(3) The following is an example of the completed transmittal form.



**TRANSMITTAL FORM FOR USE IN SHIPMENT OF TABULATING DATA**

1. ADDRESSEE
VA Data Processing Center (200/392A)
1615 East Woodward Street
Austin, TX. 78772
ATTN: Agent Orange Clerk

2. STATION NAME AND ADDRESS
VA Medical Center
50 Irving Street, N.W.
Washington, D.C. 20422

3. REPLY REFERENCE (Sta. no./symbol) 688/136B

4. EFFECTIVE DATE OF DATA

5. NO. OF PACKAGES

6. DISPATCH DATE

6A. FINAL BATCH (Check)

7. OFFICIAL RESPONSIBLE FOR SHIPMENT (Name, title and signature)
Jane Smith 389-5412

8. TABULATING DATA

| REPORTS CONTROL SYMBOL (A) | JOB NUMBER (B) | | DESCRIPTION (C) | NO. OF COPIES OF REPORTS (D) | CARD COUNT (E) |
|---|---|---|---|---|---|
| | 10 | 20A1 | AGENT ORANGE | | |
| | | | Facility number 688 | | |
| | | | Batch number 002 | | |
| | | | Code sheet count 025 | | |
| | | | | | |
| | | | | | |
| | | | | | |

9. REMARKS

VA FORM MAR 1972 **30-7252**

EXISTING STOCK OF VA FORM 30-7252, MAR 1966, WILL BE USED.

(4) The encircled area on the copy of the transmittal sheet (the address of the DPC, address of the transmitting facility, station number, and mail symbol, job number, "Agent Orange," facility number, batch number, and code sheet count) should be overprinted on the VA Form 30-7252 at each facility. The facility number entered has to be the PTF or OPC number that is coded on the code sheet.

c. Control log:

As batches are prepared for submission to the DPC an entry should be made on the control log. Instructions for the use of the control log and an example of the control log follows:

FACILITY (1) AGENT ORANGE CONTROL LOG

| (2) Batch Number | (3) # Code Sheets | (4) Date Sent | (5) Date Returned | | | |
|---|---|---|---|---|---|---|
| 002 | 25 | 10-6-81 | 10-26-81 | | | |
| | | | | | | |
| | | | | | | |
| | | | | | | |
| | | | | | | |

(1) An Agent Orange address control log should be maintained for each facility (Facility Code Number).

(2) The batch number will be assigned sequentially by facility. The batch number will be recorded on the log and on the transmittal sheet (Item 8).

(3) The number of code sheets in the batch will be recorded on the log and on the transmittal sheet (Item 8).

(4) Date the batch was mailed to the DPC.

(5) Date the batch and associated edit output was returned from the DPC.

d. Mailing:

(1) The facilities will establish their own control over the mailing of code sheets to the DPC. In order to ensure that the computer files are current, each facility should submit input at least once each month.

(2) The mailing address for the DPC is:

VA DATA PROCESSING CENTER (200/392A)
1615 East Woodward Street
Austin, TX 78772
ATTN: AGENT ORANGE CLERK

(3) Contact the Agent Orange Clerk at the DPC regarding questions about submitting code sheets, batch control, etc., The telephone number is FTS 770-7281. It is not appropriate to call the DPC in regard to questions on code sheet completion or correction of rejections. These questions should be referred to Nancy Howard, VACO, FTS 389-5412.

e. Processing:

(1) The DPC will keypunch the data from the code sheets and the records twice each month (10th and 25th). Subsequent to editing, the DPC will return all batches and the edit lists to the transmitting facility.

(2) While all code sheets will be returned to the transmitting facility, computer listings will reflect only rejected records. For correction of the rejected records, refer to the coding instructions in Attachment C. There will be no published edit list of how to correct errors; carefully following the instructions and double checking the information coded is absolutely essential! Corrections are to be made on the returned code sheet with RED pen or RED felt-tipped pen or a new code sheet can be made with the corrections in the appropriate field(s). If a new code sheet is prepared for the return of a correction, do not just complete the corrected field(s)—all of the fields must be completed as if it were an initial input. DO NOT leave any blank fields.

(3) All returned code sheets should be disposed of only after all information input is verified as correct. Refer to the RCS 10-1 schedule under Medical Administration Service section for the disposition schedule.

(4) There has been a programming change in the editing process for the Agent Orange Registry. Several new error messages are appearing. Examples of the new messages and the corrective action needed are listed below:

(a) ****** - means an error is in a field.
ACTION - correct error and resubmit.

(b) Duplicate Initial Exam - means there already appears on the file an initial Agent Orange examination for this veteran. It may be for the same reporting facility or a different VA facility.
ACTION - No action is necessary.

(c) Warning - No Initial Exam - means the file does not contain an Initial Exam record for this veteran, but the registry has accepted the follow-up record that was previously submitted.
ACTION - Reconstruct the initial exam record and submit to the DPC. DO NOT resubmit the follow-up exam.

19. A monthly statistical report will be sent to VACO, Agent Orange Projects Office, Attn: Nancy L. Howard, RRA (10A7A, Rm, 848). Do not enclose this letter with the medical record documentation and code sheets sent to 10A7, Rm. B-67. This statistical report will be prepared on a monthly basis and should arrive in VACO (10A7) by the tenth workday following the end of the month. Negative reports are required. Please assure that accurate statistics are reported. For Satellite OPC's performing Agent Orange examinations do not submit the statistical report separately. The totals should be combined with the parent facility totals. Do not use the mailing schedule described in paragraph 17 for submission of this statistical report. This transmittal letter should contain:

a. The number of initial examinations performed during the month;

b. The cumulative total of initial examinations performed;

c. The number of follow-up examinations performed during the month;

d. The cumulative total of follow-up examinations performed;

e. The number of initial examinations pending beyond the end of the month;

f. The number of veterans failing to keep an initial examination appointment.

See definitions in Attachment D for an explanation of the monthly report terminology. A copy of the format for the statistical report is illustrated in Attachment E.

It should be noted that the pending examination total (e) and the number of veterans failing to keep an initial examination appointment (f) are not cumulative totals. These apply to the report month only.

20. Special care should be addressed to the completion of the examination code sheets. A black ball-point pen or a black felt-tipped pen should be used. No pencils or blue ink pens should be used as these markings do not reproduce clearly. Carefully follow the instructions for completing the code sheets to assure that all data fields are completed. It is recommended that the Chief, Medical Information Section, be given the responsibility for the coding, completing, mailing of the code sheets to the DPC and the correcting of the code sheets to assure all areas are completed accurately.

21. This circular rescinds DM&S circulars:

10-80-203, dated September 12, 1980;
10-81-12, dated January 15, 1981;
10-81-54, dated March 19, 1981;
10-81-82, dated April 28, 1981;
10-81-115, dated June 4, 1981;
10-81-263, dated December 1, 1981;
10-82-5, dated January 18, 1982; and
10-82-110, dated June 28, 1982.

W. J. JACOBY, JR., M.D.
Deputy Chief Medical Director

DISTRIBUTION: COB: (10) only plus (10A7) 500
SS (10A7) FLD: MA-5 each and RD, DO, OC & OCRO-2 each plus 200-8
EX: Box 44-6, Boxes 60, 54, 52-1 ea. & 63-5

ATTACHMENT A

(FACILITY LETTERHEAD)

Positive Findings -- Recommended Format

Dear Veteran:

We sincerely appreciate your recent participation in the Veterans Administration's Agent Orange Registry. This effort should prove to be very helpful in assisting us to better serve veterans, such as yourself, who are concerned about the possible adverse health effects of exposure to Agent Orange.

A review of the results of your examination indicates that ________
(Use this space to briefly describe any positive findings.)________
______________________________________________________________

In view of the above findings, we suggest that you contact the Outpatient Admissions Office at extension __________ to schedule a follow-up examination. This will provide us with an opportunity to personally discuss these findings with you and to suggest or provide any essential medical treatment.

Again, your participation in the registry is appreciated.

Sincerely,

(NAME)
Environmental Physician

ATTACHMENT B

(FACILITY LETTERHEAD)

Negative Findings -- Recommended Format

Dear Veteran:

We sincerely appreciate your recent participation in the Veterans Administration's Agent Orange Registry. This effort should prove to be very helpful in assisting us to better serve you and your fellow veterans who are concerned about the possible adverse health effects of exposure to Agent Orange.

The results of your examination and laboratory tests suggest that you are presently in good health and that you have no reason at this time to be concerned about possible adverse health effects resulting from exposure to Agent Orange. However, if in the future you have a medical condition about which you are concerned, I would encourage you to seek the help and advice of your nearest Veterans Administration Medical Center.

The results of your examination will be maintained by the Veterans Administration and will be available for future use as needed.

Again, your participation in the registry is appreciated.

Sincerely,

(NAME)
Environmental Physician

Attachment C

INSTRUCTIONS FOR ITEMS 1-20 for the Agent Orange Registry Codesheet. (VAF 10-9009).

Item 1 - Facility Number - Suffix - Enter PTF facility code. Use the AMIS Suffix (BY, BZ etc) to indicate your satellite facility. DO NOT USE Q,R,S,

Item 2 - Veteran's Name

Beginning in block 8, enter veteran's last name (please print) using one letter per block. Apostrophes and hyphens in the name should not be used and empty blocks must not be left between the letters of the last name. Do not skip a space or use a comma if the last name is followed with JR, SR, I, II or III, etc.

Beginning in block 34, print the first name, one letter per block. If there is a middle name, enter the middle name beginning in block 49 - otherwise leave this block blank.

Item 5 - Type of Exam - Enter A = initial; C = follow-up. To delete an entire initial examination with a noted error after it has been accepted into the registry, resubmit the original code sheet with a "B" coded in block 59 and submit a code sheet with the correct information with an "A coded in block 59. All fields must be completed on a resubmission. The code sheets can be shipped in the same batch. An example for this usage will be for incorrect spelling of the name, incorrect social security number, changing of address etc.

To delete an entire follow-up examination with a noted error after it has been accepted into the registry, resubmit the original code sheet with a "D" coded in block 59 and submit a code sheet with the correct information with a "C" coded in block 59.

Item 6 - Social Security Number

Block 60 should be left blank. Enter the SSN in blocks 61 through 69. If the veteran does not have a social security number, place the letter "P" in block 60 and assign a pseudo SSN. (See PTF instructions for pseudo SSN). Numerical zeros must be slashed (Ø).

Item 7 - Service Serial Number

Enter the Service Serial Number beginning in block 70, unused blocks remain blank. Numerical zeros must be slashed (Ø).

If the serial number begins with US, blocks 72-79 must contain a number(s).

Fill unused block(s) with zero(s) for this instance only.

If the serial number is unknown, enter a U in block 70. Unused blocks remain blank.

Example:

| 70 | 71 | 72 | 73 | 74 | 75 | 76 | 77 | 78 | 79 |
|---|---|---|---|---|---|---|---|---|---|
| 7 | 0 | 8 | 0 | 0 | 0 |  |  |  |  |

Service Serial Number
708000

| 70 | 71 | 72 | 73 | 74 | 75 | 76 | 77 | 78 | 79 |
|---|---|---|---|---|---|---|---|---|---|
| U | S | 6 | 6 | 7 | 0 | 0 | 0 | 0 | 0 |

Service Serial Number:
US 66700000

| 70 | 71 | 72 | 73 | 74 | 75 | 76 | 77 | 78 | 79 |
|---|---|---|---|---|---|---|---|---|---|
| U |  |  |  |  |  |  |  |  |  |

Veteran does not know serial number

Item 8 - Date of Birth

Enter the numerical equivalent for the month (blocks 80-81) and day (blocks 82-83). Enter the last two digits of the year of birth in blocks 84 and 85. Numerical zeros must be slashed (0).

Example:

| MO. |  | DAY |  | YR. |  |
|---|---|---|---|---|---|
| 80 | 81 | 82 | 83 | 84 | 85 |
| 0 | 5 | 0 | 9 | 4 | 7 |

May 9, 1947

Item 9 - Current Address

Print the veteran's current address in the spaces provided. Use of one block per letter or number. Leave one blank space between street number and name. Print street address in blocks 86-111. Print city or town in blocks 112-137. Print zip code in blocks 138-142. Blocks 143-146 will be blank. Using the PTF codes, assign the proper country and state codes in block 147-151.

Item 10 - Race/ Ethnicity

Enter the appropriate code in block 152.

Item 11-13 - Sex, Martial Status & Current Status of Veteran

Enter appropriate codes.

Item 14 - Branch of Service

Enter appropriate codes in block 156.

If veteran was in more than one branch of service (item 14), code the latest Vietnam service.

Item 15 - Enter the appropriate code for Vietnam service in block 157. If the veteran did not serve in Vietnam, blocks 158-173 should be left blank.

Item 15A - Code the numerical equivalent of the month and code the last two digits of the year of last period of service in Vietnam. Numerical zeros must be slashed (Ø).

Item 15B - If veteran had two or more periods of service in Vietnam, the next to last period of service should be coded in the blocks provided.If only one period of service in Vietnam code this in 17(a) and leave 17(b) blank. Numerical zeros must be slashed (Ø).

Item 16 - Corps or Area Served

Enter the appropriate code (in block 174) for the corps or area in which veteran served. If he served in more than one, use code 6.

Item 17 - Military Unit

Enter the military unit in which the veteran served. Please specify complete unabbreviated title. (Company, battalion, corps, ship, division).

Item 18 - Last Two Periods of Service

Code the month and year of the last two periods of service in 18(a) and (b) regardless of whether or not they were in Vietnam. If veteran did not have more than one period of service, leave (b) blank.

Item 19(a) - (e) Exposure to Agent Orange

Place the most appropriate code that describes veterans exposure to Agent Orange in the block provided. Do not leave any block blank.

Item 20 - Veteran's Health

Enter the code that most appropriately describes veterans health.

INSTRUCTIONS FOR ITEMS 21-34 (THE ITEMS TO BE COMPLETED BY THE EXAMINING PHYSICIAN OR THE DESIGNATED PHYSICIAN)

Item 21 - Date of Exam

Enter the numerical equivalent of the month, day, and year in the appropriate blocks.

Item 22 - Veteran's Complaint(s)

Print the veteran's complaint(s) in the blanks provided. MAS personnel will fill in the blocks for 22(a), (b) and (c), utilizing the ICD-9-CM coding systems. Use the symptoms and signs categories (780-789) for this coding. The "78" has been preprinted for you. For uncodable symptoms, use 78999. For no known complaints use 78000.

Item 23 - Chief Complaint

Enter appropriate code (1 or 2) in the block.

Item 24 - Number of Complaints

Enter the number of complaints the veteran has in the block provided. If the veteran has 5 or more complaints, enter 5 in the block.

Item 25 - Evidence of Birth Defects in Veteran's Children?

Enter the appropriate code for item 25.

Item 26 - Diagnostic Work-up and/or Consultation

If no work-up and no consultation has been done, enter code 1 in the blocks provided. If a work-up and/or consultation has been done, enter the appropriate code (2,3,4) in the blocks provided. All blocks must have one entry.

Item 27 - Additional Workups/Consultations

Specify any additional workups/consultations performed but not listed in item 26.

Item 28 - Diagnosis

Print the veteran's major medical diagnosis(es) in the spaces provided (a,b,c). For each diagnosis listed, MAS will utilize the ICD-9-CM coding system.

Any diagnosis relating to a neoplasm should be documented in item 29.

Item 29 - Evidence of Neoplasia

Block 240 of ths section must be completed with the appropriate response. If the veteran has a neoplasm or has a known history of a neoplasm, document the appropriate diagnosis and the specific ICD-9-CM diagnostic code must be listed in blocks 241 to 245. If no neoplasm is recorded, leave blank.

Item 30 - No Disease Found

If no disease is found, put a 1 in block 246. Otherwise, leave this block blank.

Item 31 - Years of Onset

For each listed diagnosis in item 28, code the last two digits of the year of onset; leave blank if year of onset is unknown.

Item 32 - Disposition

Place a code (1 or 2) in each block provided. Do not leave any of the blocks blank.

For section d in item 32, if the veteran was referred for VA outpatient care, indicate the two digit code for the clinic in the designated blocks (257-266). Refer to the Outpatient Routing List (VAF 10-2875-1) for the clinic codes to be utilized to code this section.

Item 33 - Remarks

Utilize this space for additional information.

Item 34-36 - Name and Title of Examiner

The name and title of the examiner should be printed in the space provided. The examiner should also sign his/her name.

Information to be abstracted for a follow-up examination:

Items 1 through 13 - must be completed
Items 14 through 20 - no entry
Item 21 - must be completed
Items 22 through 33 - may be blank unless you have follow-up data to report in in any of these items.
Items 34 through 36 - must be completed

PLEASE NOTE: The first time a follow-up visit is recorded on the revised code sheet for a veteran who previously received an initial exam recorded on the old code sheet, every attempt should be made to obtain and record the information to complete Items 14-20.

Attachment D

## DEFINITIONS FOR THE MONTHLY REPORT TERMINOLOGY

1. Initial Examination: First time Agent Orange examination given for the purpose of entering a Vietnam veteran into the Agent Orange Registry. The total of initial examinations given during the period of the current report (i.e., 30 initial exams given during January).

2. Cumulative Initial Examination: Includes the total number of "first-time" examinations performed by the medical facility since the beginning of the registry in 1978. Examinations performed by satellite outpatient clinics should be included in the total cumulative figure for the VA medical center of jurisdiction. Independent outpatient clinics should report in a manner similar to the VA medical centers.

3. Follow-up Examination: Any Agent Orange-related examination/visit subsequent to the initial examination.

4. Cumulative Follow-up Examination: Includes the total number of follow-up examinations performed by the medical facility since the beginning of the registry in 1978.

5. Pending Examinations: Initial Agent Orange examinations for which appointments have been scheduled beyond the end of the month.

6. Number of veterans failing to keep an initial examination appointment: number of veterans who failed to keep a scheduled appointment during the month.

-

ATTACHMENT E

EXAMPLE
(FACILITY LETTERHEAD)

Agent Orange Projects Office (10A7)
ATTN: Nancy L. Howard, RRA
VA Central Office, Rm. 848
810 Vermont Avenue, N.W.
Washington, D.C. 20420

SUBJECT: Monthly Report on Possible Exposure of Veterans to Herbicides During the Vietnam War, RCS 11-49

1. The following information is submitted for the month ending ____ ____ ______________________; facility number_______________.

    a. Total number of initial examinations performed __________

    b. Cumulative total of initial examinations performed __________

    c. Total number of follow-up examinations performed __________

    d. Cumulative total of follow-up examinations performed__________

    e. Number of pending initial examinations at the end of the month __________

    f. Number of veterans failing to keep an initial examination appointment __________

2. Comments/problems regarding pending exams:

3. The name and FTS number of the person preparing the report:

______________________________________________________

(Name)
MEDICAL CENTER DIRECTOR

# Testimony before the Subcommittee on Compensation, Pension, and Insurance of the House Committee on Veterans' Affairs on behalf of the New Jersey Agent Orange Commission

Peter C. Kahn, Ph.
April 27, 1983

Mr. Chairman and Members of the Committee:

Thank you for the opportunity to appear here today. My name is Peter Kahn. I am Associate Professor of Biochemistry at Rutgers, the State University of New Jersey, and a member of the New Jersey State Agent Orange Commission. I appear here today on behalf of the Commission. We support HR 1961, and our arguments in its favor follow. The Commission approved this testimony at its meeting of April 15, 1983.

To convey some sense of the immediacy of the problems faced by Vietnam veterans who are ill, we offer two case histories from our files. Next, we suggest two conditions that we believe should be added to those now in the bill. We then discuss the use and misuse of science in formulating public policy, and, lastly, we examine some of the moral and political issues that are involved.

Case 1: Mr. A., thirty-seven, married, three children. He was a "river-rat" in the navy from 1965 through 1967 and was discharged in 1971. His work in Vietnam involved running small craft up and down the rivers of the Mekong Delta, and he worked on a barracks ship as well. He was decorated.

Until he fell ill in 1979 he earned a confortable living as a master chef. His primary diagnosis was of cryoglobulinemia with associated vasculitis. The conditions is an unusual immunological disorder. Other problems developed as a consequence, and the aggregate result was his death late last year.

His widow now works two jobs. That and social security are all they have. The family have large debts arising from the long illness. Their oldest child, a daughter, graduates high school this year. She is a good student and wants to go to college. The American Legion has established a scholarship fund in his memory with the stipulation that his children shall have first claim on it. It is small and may not be enough.

Case 2: Mr. B., thirty-six, married, with four children. He was an aircraft mechanic before entering the army, served in Vietnam in 1968-69, and now earns his living as an auto mechanic. He did helicopter repair work while in the service, and he was in combat.

The oldest of their children, a girl, was conceived before he went to Vietnam. In her mother's words she has "no real health problems. Has exceptionally high I. Q. School psychological evaluation done - child extremely artistic, athletic, and a 'genius'." Parental pride may be showing there! The other three children, all conceived since Vietnam, have problems. One has arthritis. Another is asthmatic. All suffer from frequent respiratory infections, including bronchitis and pneumonia. Two have been hospitalized. All three have required speech therapy, and there are other problems, as well.

The financial burden of continuing care makes it necessary for Mrs. B. to work two jobs. In January of this year she contacted the Commission. It is common for our first contact with veterans to be through their wives. The men often think it useless to seek outside help. We sent her a copy of our "Vietnam Veterans' Self Help Guide," which contains a brief questionaire. Here is her reply:

January 11, 1983

Dear Commission:

I finally succeeded in getting my husband to fill out the enclosed form from your "self help guide." I have tried for years to get him to have a physical done to no avail. So finally he got so sick he asked me to make him an appointment, which I did, and on June 28, 1982, he had his physical done and many other tests. He came home from Vietnam with sores on his chest which since have enlarged and spread to other areas such as his back and shoulders. If I had known that he was eligible for a free exam at the V.A. hospital I would have pushed him to go sooner. It's only for this reason he never had a physical done - we couldn't afford it. We have very high medical expenses from our children's illnesses. We just couldn't put any more out.

The people at the V.A. hospital in Delaware were nice, but boy it's a long wait. They would not commit themselves to say anything in regard to Agent Orange except that he has nine of the ten symptoms all the other boys have.

My husband constantly have chest pains (sometimes worst than other times). Pains particularly around the main sore in the center of his chest (which by the way is three times bigger than it was when he came home from Nam with it). He has numbness in hands, arm, legs, breathing difficulties, inflammation/irritation of sores on chest, constant trouble with bowels - either constipation or diarrhea (very seldomly has normal bowel movements), occasional headaches, dizziness. He gets cold, loses feelings and becomes weak and pale looking. It frightens me to know and feel that something is wrong with him but yet all tests performed proved to be medically negative. In other words they can't medically find anything wrong. They also said (but it isn't in writing) that his appendix is enlarged "possibly" from chemical poisoning or "whatever."

I myself don't understand any of this red tape medical talk, but being married to the same man for fifteen years I can tell you something is wrong with him. When he first came home from Nam he dreamed bad dreams and many nights I would become a victim and find myself fighting him off, for he would physically fight me thinking I was Vietcong. I grew to understand and stood by him. He still dreams but it's not as bad as it first was when he came home.

I can slowly see his health is tearing down and all this talk about Agent Orange I don't understand. Why would the U. S. government spray such a deadly chemical over "Nam" knowing our boys could be harmed? I often wonder about that and now the government doesn't want to recognize our men who fought hard in Vietnam, many who died, as veterans. So its after a great deal of persuasion getting him to fill out this form I'm finally mailing it to you. He knows about the form, but he does not know I've written this letter to you.

He has another appointment tomorrow in Deleware for he still has pains in chest (in center of main sore), has numbness, weakness, and recently developed sores on his legs. His physical appearance is run down and pale looking. Yet they can't find anything wrong with him except for his exposure to chemicals in Vietnam.

So tomorrow will be another long day of waiting so they can say "nothing" is wrong; but then I wonder. Only time will tell.

I'm not much at writing letters so forgive the wording and penmanship. I hope further research would be proven to be beneficial for all boys who were exposed to the chemicals so they can be cured or helped anyway possible. I wish there was something I could do for my husband - and I guess just being by him is enough for him.

I don't know if my letter is of any importance to you but I felt I had to write.

Cordially,

Mrs. B.

Mr. B. knows of his wife's letter now, and they agreed to let it be used here. Mrs. A. also agreed to our use of material from her husband's file.

The medical conditions which we suggest adding to the bill are melioidosis and the family of cancers celled the malignant lymphomas, including the leukemias. Melioidosis is a bacterial disease. Its origin has nothing whatever to do with Agent Orange. The disease is endemic in Southeast Asia. It can take two forms: acute, which is often fatal, and chronic. Years can elapse between exposure to the bacterium and the onset of symptoms. Cases were diagnosed in troops in the field during the war. Cases have arisen in Vietnam veterans since their return, sometimes triggered by physiological stresses such as influenza. The number of cases proven to have arisen from exposure in the Western Hemisphere can be counted on the fingers of one hand. The likelihood, therefore, of a Vietnam veteran's case of the disease having originated anywhere but in Southeast Asia is almost nil. The number of cases expected to arise in veterans is small.

The evidence supporting the inclusion of the malignant lymphomas is of four kinds:

1. Lennart Hardell and his associates in Sweden, the people whose work on soft tissue sarcomas led to the current interest in those cancers, have also studied the lymphomas. A preliminary case report appeared in *The Lancet* in 1979[1] and a detailed, case-control study was published in *The British Journal of Cancer* in 1981[2]. These results indicate that persons exposed to chlorophenols and phenoxy acids had a five to six fold greater incidence of these cancers than did unexposed persons.

2. Lumberjacks, occupationally exposed to phenoxy herbicides, appear to have an increased incidence of lymphomas and leukemias.[3]

3. One study of American workers exposed to trichlorophenols found three deaths of lymphatic and hematopoietic malignancy when, for the size of the cohort, 0.88 were expected[4].

4. Dioxin is known to suppress the cell mediated immune response in animals[5]. Organ transplant patients, who receive immunosuppressive drugs for obvious reasons, have a greatly increased incidence of lymphomas[6].

The preliminary evidence is sufficiently suggestive, we believe, to justify at this time presumptive service connection pending further work. Although the research is by no means conclusive, we strongly recommend that in the context of the bill, melioidosis and the lymphomas be included.

As a scientist, I can't tell you that the medical problems of Vietnam veterans are unequivocally the result of exposure to noxious materials during service there. I think that there is an abundance of circumstantial evidence to suggest such a connection, but that is not rigorous proof. The Federal Government has generally taken the position that in the absence of such proof no action should be taken, and not only with regard to Vietnam veterans. Political considerations sometimes alter that position, but they do so only occasionally. The burden of justifying action is thus shifted to science. We are asked to produce evidence sufficient to "convict beyond reasonable doubt". Science cannot do that. There is always reasonable doubt in the evaluation of research, particularly biological research. By requiring science to do what it cannot do before you, the Government, take action, you avoid taking action. In the process science is discredited, and if that continues, the river of good it has produced will run dry.

In shifting the burden onto science, moreoever, you shift it away from its proper place, which is on you and on the rest of the Government. The issue before you is not primarily sceintific; it is political and moral. There are no technological diasters. There are, instead, failures of political will and moral courage.

The families that we have described here today are in trouble, and they represent only a few of many in such situations. The problems of the men affect their families directly. If a man cannot keep a job, or if he has one but is so often ill that he exhausts his sick leave, he suffers doubly - from the illness and from the loss of self-respect that attends his inability to support his family. How would any of us face the future with debilitating illness that doctors can neither diagnose clearly nor treat effectively? Feeling poorly, moreover, a man is less able to deal with the stresses that his frequent incapacity creates. Often his wife must work to make ends meet, and the resulting fatigue reduces her ability to cope with the increased emotional needs of her husband and of the children. The children are not immune from the results. In addition to a lack of money and continual uncertainty about the future, their parents frequently cannot provide the kind of predictable, secure family life that children need. The parents moreover, usually recognize this, and the knowledge itself adds to their difficulties. Some marriages survive these stresses, and some do not.

We submit that the ultimate dollar cost to the country of not helping these men will far exceed the dollar cost even of generous help. Help, unfortunately, is an easily identified and vulnerable single target in a budget. The lack of help now, however, will grow into a multitude of items in many budgets, federal, state, and local. Their connection to the subject of today's hearing will be obscured, but they will nonetheless be real. In addition to direct dollar costs, moreover, there are other losses of material wealth, wealth not generated by people whose vigor is sapped and whose children's vigor is not nurtured as well as it could be. To all these a dollar value could be attached. Add to them costs for which there can never by a money equivalent: damaged lives, anxiety, and, for some, despair. We can write all this off as yet another cost of war, but if we do, we allow a rip in the fabric of a nation that can ill afford them to become a spreading hole. In the long run we'll pay handsomely for that.

If you pass this bill, then some of the people who receive help as a result may do so for illnesses that are not, in fact, service connected. If you vote it down, and if it is later shown that there is a connection between exposure to environmental hazards in Vietnam and later medical problems, then you will have declined to help men who have earned it at a time when it is needed.

We of the New Jersey Commission look at the same evidence that Government finds inconclusive and find it suggestive of service connection, some of it strongly so. If we are right, and I believe that we are, inaction now means national trouble later, for in making a choice not to act you raise questions in the minds of the people of the credibility of the Government.

Thank you.

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## Occupational Health

# DO PHENOXY HERBICIDES CAUSE CANCER IN MAN?

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***Summary*** Two Swedish case-control studies have shown an increased risk of soft-tissue sarcomas in men exposed to phenoxy herbicides and chlorophenols during their application. A high incidence of soft-tissue sarcomas has also been observed among workers employed in the manufacture of these products in the U.S.A. Other studies have failed to demonstrate this association. A third case-control study in Sweden suggests that phenoxy acids and chlorophenols may also predispose to Hodgkin's disease and non-Hodgkin's lymphoma, but as yet there is little support for this theory from other sources. Further research is urgently needed to confirm or refute these associations, to define the extent of the risk (if any), and to identify the carcinogen(s).

## INTRODUCTION

PHENOXY herbicides have been produced commercially since 1944[1] and have been widely used in Western countries both in agriculture and in forestry. They have also been sold for use in gardens, although on a much smaller scale. Over the past five years evidence has emerged of an association between exposure to these compounds (in particular phenoxyacetic acids) and the occurrence of soft-tissue sarcomas and lymphomas in man. In this paper we review the evidence for such an association and consider its implications.

## PHENOXY ACIDS, CHLOROPHENOLS, AND DIOXINS
(see figure)

The relevant epidemiological data concern not only phenoxy acids but also structurally related chlorophenols and contaminants of these products, in particular dioxins.

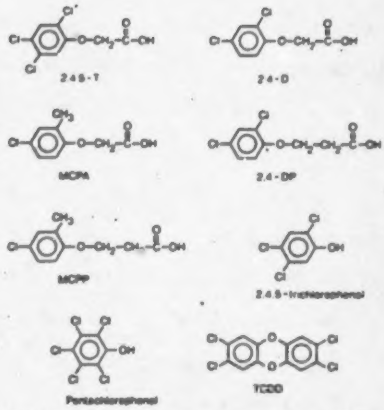
### *Phenoxy Acids*

2,4,5-T (2,4,5-trichlorophenoxyacetic acid) is used principally as a systemic herbicide for the control of woody and herbaceous weeds.[2] In the United States, which has been the major producing country, output reached a maximum of 7·9 million kg in 1968.[1] It has been manufactured in Britain since the late 1950s but has been used here on a much smaller scale than, for example, in the U.S.A., Sweden, or New Zealand. During the years 1976–80 sales for eventual use in the United Kingdom averaged 48·2 tonnes.[3]

2,4-D (2,4-dichlorophenoxyacetic acid) is used for the control of broad-leaved weeds in cereal crops and on pasture and turf.[2] Again the United States has been the world's biggest producer (36 million kg in 1968).[1] It has been made in the United Kingdom since 1950.

Both 2,4,5-T and 2,4-D were constituents of "agent orange" (a 50-50 mixture of the n-butyl esters of 2,4,5-T and 2,4-D), which was used as a defoliant during the Vietnam war.

MCPA (2 methyl-4 chlorophenoxyacetic acid), 2,4-DP (2,4-dichlorophenoxypropionic acid), and MCPP (2 methyl-4 chlorophenoxypropionic acid) are also used as herbicides.



Chemical structure of phenoxy acids, chlorophenols, and TCDD.

### *Chlorophenols*

2,4,5-trichlorophenol is an intermediate in the production of 2,4,5-T and pentachlorophenol has been used as a wood preservative since the 1930s as well as being a herbicide and defoliant.[4] 2,4,6-trichlorophenol and 2,3,4,6-tetrachlorophenol have also been used as wood preservatives.

### *Dioxins*

Commercial preparations of phenoxy acids and chlorophenols contain contaminants of which the dioxins form one group. TCDD (2,3,7,8-tetrachlorodibenzo-para-dioxin), which is found in 2,4,5-T and 2,4,5-trichlorophenol, has attracted particular attention because of its extreme toxicity. 2,4,5-T produced before 1965 contained 30 mg/kg or more TCDD,[1] but the maximum permitted limit for TCDD in products currently formulated for use in the United Kingdom is 0·01 mg/kg.[5] The dioxins which contaminate other phenoxy acids and chlorophenols are less toxic than TCDD.

## PHENOXY HERBICIDES AND SOFT-TISSUE SARCOMAS

The initial suggestion of an association between phenoxy acids and soft-tissue sarcomas came in 1977 with a report from Sweden of seven cases in men who had been exposed to phenoxy herbicides.[6] This finding prompted two studies in which the exposure of patients with these tumours to phenoxy acids and chlorophenols was compared with that of controls.

The first study was carried out in northern Sweden, where the original cases had been observed and where forestry is a major source of employment.[7] Fifty-two male patients were identified from the admission records of an oncology unit during the years 1970–77. Each of these patients was ascribed four controls matched for age, sex, place of residence, vital status (living or dead), and (in the case of patients who had died) year of death. Occupational and exposure histories were obtained from the subjects themselves, or if they had died from their next of kin, by means of a postal questionnaire supplemented by "blind" telephone interview. The estimated relative risk for phenoxy acids (most men had been exposed to both of the phenoxyacetic acids 2,4,5-T and 2,4-D) was 5·3 (95%

confidence limits 2·4–11·5), whereas that for chlorophenols was 6·6 (95% confidence limits [calculated by us] 2·1–20·9).

The second study, which was carried out in southern Sweden, where herbicides are used for agricultural work, took a similar form except that the 110 cases were identified through a tumour registry and controls were matched in a ratio of 2:1.[8] Again an association was found between soft-tissue sarcomas and exposure to phenoxy acids and chlorophenols. For phenoxy acids alone the relative risk was 6·8 (2·6–17·3) and for chlorophenols alone 3·3 (95% confidence limits 1·3–8·1). The risk ratio relating to phenoxy acids considered free from TCDD impurities (2,4-D, MCPA, 2,4-DP, and MCPP) was 4·2 (95% confidence limits [calculated by us] 1·2–14·9).

Other studies of workers exposed to phenoxy herbicides during their application have so far failed to confirm this association. However, in most cases the design of these investigations was such that only very high relative risks for soft-tissue sarcoma were likely to be detected. No soft-tissue sarcomas were reported in a study of Finnish herbicide sprayers, but the number of men followed was small and a fivefold increase in risk might easily have been missed by chance.[9] Similarly, the absence of any cases in a follow-up study of Swedish foresters and forestry foremen could well have been due to chance, since the expected number of soft-tissue sarcomas was estimated to be only 0·1.[10] Again, there were no soft-tissue sarcomas in a study of Swedish railroad workers exposed to phenoxy herbicides, but the total number of cancers found in these men was only 16.[11]

Of greater importance is a preliminary report of a case-control study of soft-tissue sarcomas in New Zealand which showed no higher risk in farmers, foresters, and fishermen than in men with other occupations (A. H. Smith, D. O. Fisher, N. Pearce, C. A. Teague, unpublished). Results of further inquiries about the exposure of patients and controls to phenoxy herbicides are awaited. Like Sweden, New Zealand has been a heavy consumer of these preparations,[12] but it is possible that in New Zealand a greater proportion of spraying has been from the air.

The other source of evidence for a risk of soft-tissue sarcomas attributable to phenoxy acids and related compounds is a series of studies of industrial workers exposed to these substances during manufacture. Because the incidence of soft-tissue sarcomas in the general population is low, individual investigations have been too small to detect any definitely increased risk. However, a review of four studies from the United States showed that 3 of 105 deaths had been due to soft-tissue sarcomas.[13] This compares with 0·07% of deaths in the total population of US males aged 20–84. Three of the four studies consisted, respectively, of 121 men in whom chloracne (an acneiform rash caused by TCDD and other chlorinated organic compounds) developed following a trichlorophenol process accident in 1949,[14] 61 trichlorophenol production workers involved in a chloracne incident in 1964,[15] and 204 employees exposed to 2,4,5-T during its manufacture between 1950 and 1971.[16] Full details of the fourth study have not yet been published. The sarcomas which caused the three deaths were a malignant fibrous histiocytoma, a fibrosarcoma, and a liposarcoma.

A fourth (non-fatal) case of soft-tissue sarcoma has now been reported in one of these groups of men.[17] In this instance the tumour was a malignant fibrous histiocytoma.

Another employee exposed to 2,4,5-T while working for one of the firms concerned in these studies has recently died of a neurogenic sarcoma, but it is unclear whether he was under observation as part of one of the studies mentioned above.[18]

In Europe three studies of workers employed in the manufacture of 2,4,5-trichlorophenol or 2,4,5-T have been described, but none of the 35 deaths reported was due to soft-tissue sarcomas.[19-21]

## PHENOXY HERBICIDES AND LYMPHOMAS

In 1979 a report appeared from Sweden of 17 men with histiocytic lymphoma, of whom 11 had been exposed to phenoxy acids or chlorophenols.[22] This led to a case-control study of Hodgkin's disease and non-Hodgkin's lymphoma (NHL) along the lines of the Swedish studies described above.[23] The patients were men admitted to the Department of Oncology in Umea, northern Sweden, during the years 1974–78 with verified malignant lymphoma. 60 had Hodgkin's disease, 105 had NHL, and 4 tumours could not be classified. Matched controls were selected in a ratio of 2:1, and exposures were ascertained by means of postal questionnaires supplemented by telephone interviews.

36·1% of patients and 9·6% of controls had been exposed to phenoxy acids or chlorophenols, giving a relative risk of 6·0 (95% confidence limits 3·7–9·7). For chlorophenols there appeared to be a dose-response relationship with relative risks of 8·4 for high-grade exposure and 2·9 for low-grade exposure. These associations applied to both Hodgkin's disease and NHL.

At present the only other direct evidence relating these products to the occurrence of lymphomas is a report of 2 men with NHL of the scalp (0·28 NHL expected) among 158 operatives in a British plant making pentachlorophenol.[24] Both these workers had been exposed to a range of other chemicals including benzene.

In addition, several studies have shown an increased incidence of Hodgkin's disease in lumberjacks and woodworkers, which could be the result of their exposure to preservatives such as pentachlorophenol.[25-27]

In the study of Swedish railroad workers 1 case of Hodgkin's disease occurred among the group of men with exposure to phenoxy acids, but the expected number of lymphomas was not given.[11]

## DISCUSSION

In the Swedish case-control studies steps were taken to exclude the possible confounding effects of exposure to other pesticides and chemicals. Their main weakness lies in the possibility of bias resulting from differences in the recall of distant events, particularly between patients and healthy controls. These differences may have been exaggerated by suggestion during previous interviews in hospital and by the effects of newspaper publicity on patients and their relatives. The possibility of unconscious bias on the part of the interviewer must also be considered, since in studies of this type it is virtually impossible for an interviewer to remain "blind". That positive associations were found for each of the three cancers studied (soft-tissue sarcomas, NHL, and Hodgkin's disease) is curious. The most obvious explanation is that the method in all three studies is subject to a serious undetected bias. However, it should be borne in mind that the tissues in which these tumours occur are embryonically related, which lends some credibility to the occurrence of a common aetiological factor.

Some of the defects of the earlier studies have been remedied in a further report by one of the Swedish authors.[28]

An analysis limited to the data obtained with self-administered questionnaires before interview produced only minor changes in the estimated risk ratios. Also, comparison of the same cases with a different control group comprised of patients with colonic cancer, whose recollection of previous exposures is likely to have been more comparable with that of the cases, still suggested a five-fold increase in the risk of soft-tissue sarcoma after exposure to phenoxy acids and chlorophenols.

It seems likely that many of the workers included in the American cohort studies have been exposed to other chemicals as well as trichlorophenol or 2,4,5-T. Also, the number of sarcomas observed in these investigations has been small. On the other hand the classification of men as exposed to trichlorophenol or to 2,4,5-T depended on data recorded before the occurrence of the tumours and is not therefore subject to biases of recollection.

On their own neither the Swedish nor the American studies provide convincing evidence that the incidence of soft-tissue sarcomas is increased after exposure to phenoxy acids and chlorophenols. However, when they are considered together the whole becomes more persuasive. The risk does not appear to be specific for any single type of sarcoma and, as has been noted above, it is surprising that the association should apply to tumours of such a variety of tissues. On the other hand, so little is yet known about carcinogenic mechanisms that it would be unwise to discount the empirical evidence on theoretical grounds.

That pesticides containing phenoxy acids (in particular phenoxyacetic acids) and chlorophenols are associated with soft-tissue sarcomas (and perhaps other tumours) would not necessarily imply that these compounds are themselves carcinogenic. The effect might be due to contaminants such as the dioxins. Apart from its well-documented acute toxic effects there is now mounting evidence that TCDD is carcinogenic in animals. An increased incidence of hepatocellular carcinomas and squamous carcinomas of the lung and upper airways has been described in rats fed TCDD,[29] and in another study in rats tumours of a variety of organs were produced.[30] Experimental data concerning other dioxins and phenoxy acids are limited.

Almost all studies in man relate to populations exposed to phenoxy acids and chlorophenols contaminated with TCDD. The Swedish data suggest that the increased risk of soft-tissue sarcomas applies to phenoxy acids free from TCDD impurities, but in a retrospective study in which the exposures occurred many years before the onset of the tumours it is difficult to be sure that no exposure to TCDD occurred.

On the present evidence it seems possible that soft-tissue sarcomas have arisen in association with exposure to phenoxy herbicides during both manufacture and use in the field. There is insufficient information at present to determine whether phenoxy acids themselves, TCDD, or some other contaminant is the cause. If TCDD is the carcinogen, increasingly stringent regulation of dioxin impurities may already have eliminated the hazard in the United Kingdom.

It is as yet impossible to estimate with any precision the risk of soft-tissue sarcoma due to phenoxy herbicides. The Swedish work on men exposed during their use suggests a relative risk of about 5, whereas American studies of workers exposed in manufacture point to a higher value. This disparity could be due to differences in dose.

Over the past 30 years phenoxy herbicides have made a valuable contribution to agriculture and forestry. Experience of other herbicides which might be used as alternatives is limited, and further information is therefore urgently needed to confirm the associations which we have discussed, to identify the underlying carcinogen, and to define more precisely the extent of the risk. TCDD can now be assayed in blood years after exposure, and this may prove a valuable tool in future investigations.[31] In the meantime there is suggestive evidence of a biological association between phenoxy herbicides (or their contaminants) and soft-tissue sarcomas. The evidence relating these products to the occurrence of lymphomas is weaker.

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Stanley N. Zwaik
NATIONAL COMMANDER

April 25, 1983

The Honorable Douglas Applegate
Chairman, Subcommittee on Compensation
Pension, and Insurance
House Committee on Veterans' Affairs
335 Cannon House Office Building
Washington, DC 20515

Dear Mr. Chairman:

I regret that we will be unable to testify before your Subcommittee on April 26th, in regard to legislation authorizing a presumption of service-connection for disabilities linked to exposure to dioxin-laced herbicides (such as Agent Orange) in Vietnam. However, I would appreciate it if this letter could be entered into the formal hearing record.

As we stated in our testimony before the House Committee on Veterans' Affairs on March 17th, we support such legislation. Currently, Vietnam veterans are being told that they must wait to be compensated for such disabilities, until long-delayed federal studies of the effects of this exposure are completed. Meanwhile, the Environmental Protection Agency is purchasing all of the homes in Times Beach, Missouri, due to concern about the effects on that city's inhabitants of exposure to dioxin-laced waste. To help those in Times Beach now, while continuing to deny compensation to Vietnam veterans, creates a double standard to the veterans' disadvantage.

Therefore, we support H.R. 1961, introduced by Rep. Thomas Daschle. Independent scientific evidence already shows links between the three diseases targeted for compensation by H.R. 1961 and exposure to dioxin-laced herbicides such as those used in Vietnam. Further, we note with approval that this bill would allow the V.A. to provide compensation for additional disabilities as links are found between these diseases and herbicide exposure.

We also urge the rapid completion of the pending Federal studies of the effects of herbicide exposure on Vietnam veterans to ascertain whether other medical conditions warrant compensation.

While the impact of H.R. 1961 would be relatively limited, we believe that passage of this bill would be a major first step towards addressing the needs and concerns of Vietnam veterans.

Sincerely,

Stanley N. Zwaik

Stanley N. Zwaik
National Commander

VIETNAM VETERANS AGENT ORANGE VICTIMS INC. (AOVI)
27 Washington Square North
New York, New York 10011
212-460-5770
Frank McCarthy
President

April 19, 1983

Subcommittee on Compensation
Pension and Insurance
Att: Douglas Applegate, Chairman
Committee on Veterans Affairs

Mr. Chairman, members of the Subcommittee, ladies and gentlemen:

I wish to present evidence, both written and oral, as to the validity, necessity, and urgency of implementing HR 1961, "The Vietnam Veterans Agent Orange Relief Act."

As to the validity, I have enclosed various medical, scientific, and public literature to substantiate the fact that, indeed, too many Vietnam Veterans are effected with soft-tissue sarcomas, porphyria, chloracne, and much, much more.

However, no credible statistical data as to the correct amount of Veterans effected or suffering ill health problems are available from the VA or any other U.S. Agency. Many State and Federal agencies as well as independent groups such as V.V.A.O.V.I., are presently engaged in processing and publishing such data. Hopefully, within 1983 our national statistical processing will be made available to this committee and the nation.

HR 1961 is designed, I am sure, to provide a long awaited and necessary step towards providing relief to the Veterans effected with the illnesses previously stated. A first step, if you will, by the Congressional leadership of this nation towards addressing the illnesses commonly known and associated with exposure to Agent Orange and Dioxin.

The association between exposure to Dioxin and the ill health effects of soft-tissue sarcomas, porphyria, and chloracne is demonstrated, not only in the enclosed literature, but in exposed forest workers, laboratory workers, railroad workers, animal experiments and Vietnam Veterans. I will not attempt to go into detail with this scientific literature as you will receive expert testimony on this subject tomorrow.

However, the necessity to enact HR 1961 is not only of the utmost importance, but increases with each diagnosis of cancer and with each day that a cure for chloracne is not found.

Many people within our government feel that all that can be done for the Veterans is being done via medical and scientific studies.

The fact is, out of the approximately 108 studies currently being developed or initiated not one single study is designed to find a positive treatment or cure for chloracne. Vietnam Veterans have been effected with this disfiguring skin disease ever since they entered the defoliated areas of Vietnam. It is common knowledge, now, throughout the medical and scientific communities of the world that chloracne is caused by exposure to Agent Orange and dioxin! Paul F. Oreffice, the President of Dow Chemical Company, even stated last month on the "Today" show that dioxin causes chloracne.

It is possible that some Veterans contracted chloracne since returning from Vietnam, however, the majority have suffered from this illness for years. The U.S. did not send any sick men to fight in Vietnam. As a combat Veteran I know, as do all 2.7 million Vietnam Veterans, that the average age of the Veterans was 20 years old, and that all of us were medically scrutinized and determined to be combat ready and physically fit before we were ever even sent to Vietnam.

Mr. Chairman, members of the committee, I say to you again, "there is no treatment or cure for chloracne!" Dow and the other chemical companies do not even have a cure for chloracne for their workers suffering from it. Vietnam Veterans are suffering from chloracne! The very least you can do is to support a study designed to create a treatment or a cure for chloracne.

The daily existance of those Veterans effected with soft-tissue sarcomas is the ultimate in peril. Most are terminal and their families and friends are engulfed in anguish. There is no greater tragedy than young men dying in the prime of their lives. They are fighting not for compensation but to live another day. Another day with their wives, parents, children and friends.

Men like V.V.A.O.V.I. founder Paul Reutershan, V.V.A.O.V.I. vice-president Edmund P. Juteau, and V.V.A.O.V.I. vice-president Charles Hartz III, died of soft-tissue sarcomas. They died with their lives open to public scrutiny, with their medical case histories open for the world to see and dared anyone to refute the fact that their cancer was caused by their exposure to Dioxin. No one can!

They and many other Veterans of the Vietnam war died "getting the word out" as to the toxicity of Dioxin/Agent Orange. They died helping thousands of other Veterans suffering and dying

who constantly come to V.V.A.O.V.I. for help
The least you can do is support what they died for and compensate all those others so unfortunate as to die as they did.

It must be noted that they died so that the people of Times Beach, Mo. would not have to walk back into their Dioxin contaminated homes.

I was told by a representative of this committee that some of you are not convinced that there is an association between chloracne, soft-tissue sarcomas, porphyria with exposure to Agent Orange and Dioxin. It is not difficult to determine which of you are not convinced. You fit into the category either of not reading the existing literature [medical and scientific] on the health effects of exposure to these chemicals outside of that of the chemical companies, or, into the category of having very little experience with Vietnam Veterans and their families.

I'm sure of this because if you had read the literature and have seen the Veterans, you would no longer need to be convinced. You would react as do all intelligent, aware and compassionate Americans -- with humility and sympathy.

I wish to inform this committee of the fact that the major Federal Agent Orange class action law suit will go to trial on June 27, 1983. Although our organization is not a party to the suit, our founder, Paul Reutershan, initiated the suit and V.V.A.O.V.I. has sought out hundreds of attornies to represent the Veterans and their families. The V.V.A.O.V.I. American flag flies proudly in the courtroom.

This suit relates to HR 1961 in that the largest body of information existing in the world pertaining to Agent Orange, Dioxin and possible ill health effects from exposure is being examined by the court, the chemical companies and by the Veterans attornies. The illnesses pertaining to HR 1961 are but a few illnesses claimed by the Veterans and their attornies. However, they are three of the most crucial.

I submit to you, although I am bound by order of the court not to make public certain information, I can say and I am, indeed, compelled to say that the controversy surrounding the Agent Orange/Dioxin issue will be exposed to public scrutiny in this suit. The matter of whether Agent Orange/ Dioxin causes the ill health effects claimed by the Veterans isn't even being fought and may not be fought if the chemical companies settle out of court. However, the can of worms is opened and soon all of the nation shall know who knew what and when. This committee must now demonstrate their responsibility to the Veterans in HR 1961 before the dirt is slung. The national media is also preparing to shed light on the issue and soon.

All of this shall come to pass regardless of who is at fault. It is a result of the disclosure of previously hidden information, the passing of time and most of all, the unavoidable demand of history. This committee has the unique time and place in this history to show the people of Times Beach and the Vietnam Vets that you took up that responsibility and that you care!

For those of you in this committee who are not yet convinced, please take note of the first report of the American Medical Association's scientific council report on Agent Orange and Dioxin. I wish to quote their recommendations:

" All physicians be alerted through AMA publications to the classical signs of chloracne and the possible signs and adverse effects of Dioxin exposure. They (doctors) should be encouraged to enlist in the present efforts to identify and treat those persons who have had serious exposures to Dioxin, and to cooperate in the collection of vital information that is needed for the ongoing human epidemiologic studies."

Members of the committee, I implore you to listen to the AMA and please know that the vast majority of medical personnel who read their publications will be looking for chloracne and other possible adverse effects which are being manifested in the health of Vietnam Veterans.

Further, and more importantly, the enclosed article published in the New England Journal of Medicine substantiates the fact that doctors are following the AMA's recommendations. This article is, in effect, a medical testimony of the fact that the "Interagency Work Group" is stating to the medical and scientific community that, "there has been no excess incidence of cancer of any kind, " pertaining to Vietnam Veterans and that "follow-up of the Veterans and their families to date has not revealed any serious untoward effects of such exposure, except for chloracne."

To this committee our organization hereby charges the Interagency Work Group with the following: making such false and damaging statements pertaining to the ill health effects facing Vietnam Veterans and their families which have not and cannot be verified by credible statistical data, that they are in fact following the Dow position on Agent Orange and Dioxin. We challenge them to substantiate those statements to this committee with real and verifiable statistical data.

Those doctors at Emory University School of Medicine, P. Ravi Sarma, MD, and Julian Jacobs, MD, are not involved with our group or with the Agent Orange issue at all to our knowledge. They represent the rewards for the AMA's report and the evolution of the Agent Orange issue.

If the Interagency Work Group has data to substantiate their statements this committee as well as the Veteran population of this nation have a need and a right to know. If they do not have such data this committee should condemn such statements.

Gentlemen, it has taken this nation's political leadership more that five years to reach this point of even considering shifting the burden of proof from the Vietnam Veterans to the Veterans Administration where it belongs. Show us now that you are on the side of the Veterans and not the chemical companies.

I must say that to V.V.A.O.V.I. HR 1961 is puny compared to the need, limited in it's ability to bring justice for all Veterans effected and nonexistant in it's ability to force the VA to provide the Veterans and their children with the necessary health care services and programs. However, HR 1961 is important to all Vietnam Veterans because it is a start. It shows respect and compensates our dying and our incurable. We shall continue to fight for all of the others and we will win for them as well.

Dr. Arthur Galston, the professor at Yale University said at our national convention that the Vietnam Veteran community is a "more educated group than most in America" in terms of knowledge of Agent Orange and Dioxin. We have, indeed, learned much over the last five years. He also said speaking from his 40 years of experience on the issue, "that the chemicals used to make Agent Orange penetrate into the trees [emulsifiers] can cause the kind of damage the Vietnam Veterans are claiming."

V.V.A.O.V.I. and a greater number of Veterans have read the vast amount of existing scientific literature. We have provided one-on-one programs and services to the suffering and dying Veterans for five years. We have permitted Dr. Lenard Hardell to scan our data and he has confirmed that our Veterans are suffering from the same soft-tissue sarcomas as his forest workers in Sweden. His studies were used by the US Environmental Protection Agency to ban the major uses of 2,4,5,-T and Dioxin.

I compel this committee to read, as we have, the Annals of the New York Academy of Sciences, Vol 320, the "Health Effects of Halogenated Aromatic Hydrocarbons[Dioxin], 1979. You will read the lines as we have, "Dioxin produces a variety of Toxic effects in laboratory animals and human beings," and if you read on you will no longer need to be convinced. You must read more than the VA's "Worried About Agent Orange?"

This committee must also know that the Australian Vietnam Veterans are already receiving compensation for soft-tissue sarcomas. Our brothers in Australia have achieved this because they have judicial review in their VA system, we do not. We have you -no other entity can force the VA to give us help.

For five years V.V.A.O.V.I. has been forced to do the job of the Veterans Administration. We have been forced because the VA would not provide the necessary programs and services crucial to those effected and will not even examine our children. Why?

After five years of negative Media coverage, a series of negative Government Accounting Office [GAO] reports, the disgrace of having the inept VA study given to a credible agency such as the Center for Disease Control, the VA still continues to deny the Veterans effected with those programs. Why?

In conclusion, I wish this committee to know that the jury "is in" on Chloracne, Soft-tissue Sarcomas and Porphyria and the proof is overwhelming. HR 1961 is in your hands and history shall judge you as to how you acted upon it. The Vietnam Veterans will judge you as to your credibility, compassion and justice.

It is the opinion of our organization that you will not enact HR 1961. We base that belief on the actions of many other legislative bodies who have done nothing for the Veterans and their families effected.

Further, we base our opinion on the fact that the VA, after five years, still refuses to provide the Vietnam Veterans of this nation with the existing medical and scientific literature on the various illnesses commonly known to be associated with exposure to Agent Orange and Dioxin, regardless of any cause and effect relationship. We should receive the information because it would save lives and alleviate the suffering.

I am compelled to enter into the record our organization's belief that the Dow Chemical Company is causing and has caused great discredit upon the U.S. Airforce, the U.S. Military, the Veteran's Administration, and the U.S. government in general.

The enclosed New York Times article demonstrates the fact that Dow and the others were aware of the ill health effects from exposure to Agent Orange in 1965, and did not alert the U.S. Army or the government. From 1965 til the end of the war(1973), Dow and the others permitted the Veterans to be exposed knowing they would be damaged by the chemicals.

The Dow advisors who served in Vietnam were advising our Military to "use more, it's better." They knew throughout the war that the Veterans would return to a Veterans Administration that had no knowledge of such illnesses as Chloracne and did not inform the VA of the fact that the Veterans would suffer illnesses.

Further, these chemical companies have more important information pertaining to the ill health effects of exposure to Dioxin that is of the utmost relevance to this hearing, this legislation, and to compensation for Vietnam Veterans and their children. You must have this information!

I implore this committee to demand all existing information pertaining to Agent Orange and Dioxin from the Dow Chemical Company and all of the other chemical companies involved.

In conclusion, I must state the fact that the U.S. did not commit chemical warfare during the Vietnam war in terms of Agent Orange and Dioxin. Our military and our government is not at fault for the illnesses of the Veterans. Our nation must not permit Dow to continue to bring discredit upon our Military, our government, and our nation, for the devastation, suffering and death which resulted from their product.

I am,

F. McCarthy

Frank McCarthy, President, Vietnam Veterans Agent Orange Victims
President, Vietnam Veterans Relief Foundation
Chairman, Vietnam Veterans Unifying Group
Consultant: Connecticut State Herbicide Information Commission

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ANNALS OF THE NEW YORK ACADEMY OF SCIENCES
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# HEALTH EFFECTS OF HALOGENATED AROMATIC HYDROCARBONS

Edited by William J. Nicholson and John A. Moore

The New York Academy of Sciences
New York, New York
1979

Ann NY Acad Sci 320:275-307(1979)

# ENVIRONMENTAL CHLORACNE: UPDATE AND OVERVIEW

James S. Taylor

*Section of Industrial Dermatology*
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*The Cleveland Clinic Foundation*
*Cleveland, Ohio 44106*

Since 1974, when this author reviewed chloracne (*Chloracne—A Continuing Problem*)[1] a number of new outbreaks of the disorder have occurred, both in the United States and abroad. Additionally, the scope of the problem has expanded to include involvement of other nonindustrial populations and precipitation by brominated aromatic compounds. Thus, a more appropriate term might be environmental halogen acne or haloarylacne after the terminology of Weirich.

Since chloracne has been extensively reviewed by Crow[2] in 1970 and Weirich[3] in 1978 this report will present findings from the more recent outbreaks as well as an overview of the subject.

Chloracne is a refractory follicular dermatosis produced by environmental exposure to certain halogenated aromatic compounds, and which may be accompanied by systemic toxicity.[1]

Between 1897, when Von Bettman first observed chloracne, and World War II most cases of chloracne were caused by the chloronaphthalenes and polychlorinated diphenyls, the latter now referred to as PCBs. Since then most attention has focused on the role of trace contaminants of PCBs, herbicides, herbicide intermediates, and flame retardants, among others.[1-3] The chemicals which have been associated with chloracne are listed in TABLE 1.

## POLYHALOGENATED NAPHTHALENES

The polychloronaphthalenes have been used as boat hull coatings, because of their antimagnetic properties, in the electronics, radio, and television industries as insulating waxes, sealing compounds, and dielectrics for condensers, and also as high-pressure additives for lubricants, and as wood preservatives. Although these functions have now largely been replaced by plastics and silicones, chloracne from chlorinated naphthalenes was reported in New York State in 1972 by Kleinfeld *et al.*[4] Fifty-six cases were found in workers exposed to tetra- and pentachloronaphthalene used in the application of wax insulation to wire coils at an electric plant. Exposure by contact and inhalation resulted from inadequate exhaust ventilation and poor personal hygiene.

Crow has emphasized that of the chloronaphthalene compounds, fumes are the most potent chloracnegens; solutions are less so, and solids rarely cause chloracne.[2] Shelly and Kligman,[5] in experiments on human subjects, showed that penta- and hexachloronaphthalenes were potent acnegens, whereas tri-, tetra-, hepta- and octachloronaphthalenes were nonacnegenic. Chlorinated naphthalene oils and wax-like solids are manufactured in the United States under the trade name Halowax®.[6]

Hexachloronaphthalenes may occur as contaminants of PCBs and the polybromonaphthalenes may occur as contaminants of PBBs and may be chloracnegens in those circumstances.[7]

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# THE AGENT ORANGE MYSTERY

## EARTH

By Pamela Weintraub

Bill Singley shivered as he trudged past the ramshackle brownstones of South Broad Street. The hefty martial arts instructor made his way to the first-floor office of number 1427 and fell painfully into a chair. Every muscle depleted, each joint throbbing with Philadelphia's latest chill, he wondered whether he'd ever teach karate again.

The young internist who finally invited Singley into the bare inner office didn't look as if he could offer much hope. He simply fixed his patient with a blunt gaze and solemnly began his exam. After 30 minutes Dr. Ronald Codario could tell Singley only that his liver was bloated and his nervous system damaged.

By June 1981, Dr. Codario had Singley hospitalized for a "workup." A body scan and biopsy told him that the distraught patient's scarred liver had deteriorated like an alcoholic's, though Singley insisted he rarely drank. Then, in the following weeks, Singley's symptoms mounted: His fingers and toes grew numb; his head and abdomen throbbed; he became anxious and confused.

Codario couldn't decipher the cause of the strange illness, but he did know that Singley was a Vietnam veteran. Before long, he started wondering whether the symptoms might not be due to the notorious agent orange—a herbicide that had been dumped on Vietnam to destroy crops that fed and camouflaged enemy troops. Codario told Singley he'd probably been exposed to some kind of toxin and advised him to see a lawyer.

A few weeks later Codario received a call from Hy Mayerson, a Philadelphia attorney who represented vets with illnesses they claimed were caused by agent orange. "Would you like to see some other veterans who've had exposure?" Mayerson asked. "You might be able to help." Codario said sure, and the next day he received weeks of background reading in the mail.

Agent orange, Codario learned, was one of many herbicides sprayed in Vietnam during the 1960s and 1970s. Given its name because it was shipped in orange-striped barrels, agent orange was made largely of a chemical called phenoxyacetic acid. It was usually contaminated with a small molecule known as dioxin, said by some to be the most potent toxin on Earth. Eleven thousand veterans were suing agent orange manufacturers for what they claimed were dioxin's dire side effects: depression, loss of sex drive, joint pain, even cancer. Large numbers of their children, they charged, were being born with deformed hearts, spines, and limbs.

But the experts said there was no real evidence—no *biochemical* evidence—to support such claims. The veterans' troubles, these experts said, were nothing more than a potpourri of unrelated symptoms afflicting the general population in roughly the same proportions.

After examining about 50 or 60 veterans referred by Mayerson, however, Codario observed a strange pattern that made him think otherwise. Like Singley, veteran after veteran complained of personality changes, numbness, aching joints, and extreme sensitivity to sunlight. Amazingly, almost all of them seemed to be describing porphyria (pore-fear-ia), the rare disease that drove King George III mad during the American Revolution.

If Codario could *prove* that his patients had some form of porphyria, he would be the first researcher to establish a biochemical link between agent orange and the veterans' symptoms. With such evidence in hand, he'd be well on his way to helping them get the economic and medical aid they'd been denied for years. With a bit of probing, he might even find a cure.

Classical porphyria, Codario knew, results when the liver churns out massive quantities of chemicals known as porphyrins (pore-fur-ins). Porphyrins produced by healthy livers normally combine with iron and protein to form hemoglobin, the crucial pigment in the red blood cell. But sometimes—because of a genetic deficiency, or because of an unwanted chemical—the liver becomes diseased, manufacturing porphyrins in excess. The extra molecules then travel through the body—to the skin, where they cause huge blisters, to the stomach

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AGENT
ORANGE
KILLS

*Medical research may soon vindicate veterans who proclaim the dangers of agent orange*

The
New England
Journal of Medicine

Established in 1812 as The NEW ENGLAND JOURNAL OF MEDICINE AND SURGERY

VOLUME 306 MAY 6, 1982 NUMBER 18

## THORACIC SOFT-TISSUE SARCOMA IN VIETNAM VETERANS EXPOSED TO AGENT ORANGE

*To the Editor:* Case-control studies from Sweden report a fivefold to sixfold increased risk of soft-tissue sarcoma in lumberjacks exposed to herbicides containing phenoxyacetates.[1,2] Long-term follow-up studies in cohorts of industrial workers employed in the manufacture of these chemicals have disclosed seven cases of soft-tissue sarcoma to date.[3-5] On the basis of studies in laboratory animals,[6] it is currently thought that dioxin (2,3,7,8-tetrachlorodibenzo-*p*-dioxin), an impurity present in these chemicals, may be the carcinogen. However, more follow-up and further studies are needed to define the true risk of malignant disease and the actual carcinogenic substance or substances involved.

In Vietnam, where approximately 2 million troops were stationed for various periods between 1962 and 1971, nearly 45 million kg of Agent Orange and other defoliants were used.[6] Follow-up of the veterans and their families to date has not revealed any serious untoward effects of such exposure, except for chloracne. So far there has been no excess incidence of cancer of any kind.[6] We now report three cases of thoracic soft-tissue sarcoma in persons who served in Vietnam and were exposed to Agent Orange.

Patient 1 worked in Vietnam as a member of a helicopter-rescue unit of the United States Air Force in areas where defoliants were used during 1966 and 1967. In July 1979, at the age of 46, he underwent resection of a right upper-lobe lung lesion, which was diagnosed as inflammatory fibrous histiocytoma.[7] He died in July 1981 with progressive lung and brain lesions.

Patient 2 was a United States Marine in Vietnam during 1966 and 1967, stationed in areas where defoliants were being used. Twice he was "completely soaked with Agent Orange." In December 1979, at the age of 32, he underwent a left pneumonectomy for a mediastinal fibrosarcoma. In June 1981, pulmonary metastases developed. He is currently receiving combination chemotherapy.

Patient 3 was in the United States Army in Vietnam in 1971, serving in areas where defoliants were used. In January 1981, at the age of 31, he was evaluated for a left pleural effusion. At exploratory thoracotomy in August 1981 a pleural/diaphragmatic leiomyosarcoma was noted and resected.

Soft-tissue sarcomas are rare neoplasms. If there is a true risk of these neoplasms in veterans who served in Vietnam, follow-up studies should be able to define it. If there is an increased risk of malignant disease in these veterans, then a more critical question that has to be addressed is: Are the defoliants causative, or is some other unidentified environmental factor responsible, acting alone or in concert with the defoliants?

P. Ravi Sarma, M.D.
Julian Jacobs, M.D.
Emory University
School of Medicine
Atlanta, GA 30333

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